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ORIGINAL ARTICLES

SYMPOSIUM ON SEROUS INFLAMMATIONS.

CYTO-DIAGNOSIS OF PLEURITIC AND PERICARDIAL FLUIDS OF ALL SORTS.*

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The problem of establishing an etiologic diagnosis, in studying the inflammations of the thoracic serous cavities, by determining the cell content of the exudate, is not essentially different from its application to other endothelium-lined cavities. Although this means of diagnosis has received marked attention only since 1900, at which time the result of Widal's work appeared, yet as many as twenty years previously, work along the same line was published by Ehrlich and followed by others, among whom was Quincke. There is at present an extensive literature, a large part of the work having been done by the French and Italians. Having nothing new to offer, my endeavor will be to present, in a few words, the present status of the subject.

First, let us inquire as to how the tissues react to various stimuli. One would naturally expect to find some similarity existing between the exudates produced in various parts of the body by any one variety of bacteria, irrespective of whether this exudate is poured into a serous cavity or remains in the tissue spaces. In reality, we do find some similarity, although we must admit that at times the difference is marked enough to give rise to confusion.

Cytodiagnosis depends upon the recognition of the following elements: 1, The lymphocyte, which corresponds to the same cell in the blood; 2, the polymorphonuclear leucocyte, also corresponding to the same element in the blood; 3, the endothelial cell. The first two are the chief cellular constituents of exudates or inflammatory fluids; and the third is a prominent feature of transudates or fluids the result of mechanical disturbance.

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It is generally accepted that the polymorphonuclear leucocyte is derived from the blood, and it is practically self-evident that the endothelial cells are derived from the lining of the serous cavity or from the lymph spaces draining into it; it is necessary, therefore, to account for the origin of the lymphocyte only. The question as to whether this cell is derived from the blood or from the connective tissues, has long been controversial. In a recent publication, Orth[1] claims to have seen the lymphocyte leaving the vessel wall by means of ameboid motion; from this we must admit the possibility of these cells reaching the fluids of the pleura and pericardium. Judging from this and from analogy, there is no good reason for trying to refute the probability that the lymphocyte of the fluids under consideration is the lymphocyte of the blood. We know that this is the cell that is attracted especially by tuberculous infections, and only in case of secondary infection do we find the polymorphonuclear leucocyte. On the other hand, the latter is the one that is attracted to the tissues, as the result of the irritation of the toxins of the various pyogenic bacteria.

In case of fluid in the thoracic serous cavities, the diagnostic question is usually, "Is it a tuberculous or a non-tuberculous inflammation, or is it merely the result of mechanical hindrance to circulation?" I will, therefore, mention briefly the cellular characteristics of these conditions.

Tuberculous Exudate.—Although we find some neutrophiles (i. e., polymorphonuclear leucocytes) and endothelial cells, the one upon which we depend, if upon anything, for diagnosis, is the lymphocyte. It may be the only cell found. If of the cell aggregate over 50 per cent. are lymphocytes, it points toward tuberculosis and, in case this number is 90 per cent. or more, it throws a very strong probability in favor of it. Early in the course of a tuberculous exudate (first few days), according to many observers, the neutrophiles are in excess, to be gradually replaced by the more characteristic element. In case a tuberculous cavity of the lung communicates with the pleural cavity, the fluid, on account of the secondary infection, will exhibit many, or a preponderance of, neutrophiles. Lossen[2] claims that whether we have lymphocytes or polymorphonuclear leucocytes in a fluid depends not upon the cause of the inflammation *per se*, but upon the severity of it. J. L. Miller[3] has shown that in long-standing simple pleuritis, or in hydrothorax, there may be a preponderance of lymphocytes.

Non-Tuberculous Exudate.—It is usual to find an excess of neutrophiles. Early in the disease there may be large numbers of endothelial cells; later the percentage is reduced, either because of the disproportionate increase of the neutrophile, or because the serous membrane becomes covered with fibrin, preventing further desquamation. It is probable that the two factors are combined. There may be great difficulty in recognizing the neutrophile in long-standing exudates, as will be shown later on.

1. Orth: Deutsche Medizinische Wochenschrift, Jan. 18, 1906.
2. Lossen, J.: Deutsches Archiv. für Klinische Medizin, vol. lxxxvi, Nos. 1-3, December, 1905.
3. Miller, J. L.: American Medicine, Nov. 12, 1904.

Transudates.—The characteristic element is the endothelial cell. These cells may occur singly, or what is more important from our standpoint, in larger or smaller sheets. As these cells are always accompanied by either one or both of the two other varieties of cells mentioned, it is readily seen that it is difficult or impossible to make a diagnosis of a transudate from the cell-content of the fluid alone. Especially is this true, when we consider the fact that, in inflammatory fluids, especially early, we may find a great many endothelial cells. But, if we find these cells very numerous, especially if they occur in sheets, it is at least a hint in the direction of a transudate.

The red blood corpuscle being of no diagnostic importance, has not been considered.

It is not out of place to say a few words with reference to the technic of preparing specimens after the aspiration of the fluid. 1. Centrifuge. In case the fluid contains a very large percentage of albumin the sediment may be mixed with normal salt solution and material again centrifuged. 2. Sediment spread on slide and dried in the air. 3. Fixation. (a) Absolute alcohol and ether (equal parts), or (b) formalin 1 part, absolute alcohol 9 parts. 4. Staining. (a) Hematoxylon and eosin; (b) Wright's stain (this requiring no previous fixation).

Great difficulty may arise in recognizing the various cells, because of degenerative changes that occur during the time the fluid is in the serous cavity. For example, the neutrophiles may lose their granules, and, what is even more confusing, the elements of the nucleus may fuse. It will then resemble very closely the lymphocyte, but is apt to betray its identity by retaining an opening in the center of the nuclear mass. Lossen presents some interesting comparisons between the cells of the exudate and the cells found in the tissue at autopsy. There may be only a few neutrophiles in the tissue, when there are a great many in the fluid. There may be almost complete absence of extravascular red blood corpuscles in the tissues, whereas, in the fluid the latter usually exceed the leucocytes. In cases exhibiting even a purulent fluid in the pleural cavity, there may be found in the serosa and subserosa, on section, very few neutrophiles. In these cases Lossen found that in the tissues the majority of the cells were round, with intensely stained nuclei and small amount of perinuclear protoplasm. He offers two possible explanations for the disparity. The first is that the cells may wander so quickly through the serosa that, on section one sees very few of them, whereas the fluid exhibits the aggregate. If, at the time of exudation, the connective tissue spaces are large, that is, "loosened up," we find a collection of neutrophiles or of red corpuscles in the tissue. Based upon this fact, the second explanation he offers is that the cells with single deeply staining nuclei are the neutrophiles, which, in firm tissue, are not allowed their usual development, this being possible, however, in case tissue is "loosened up" by edema. The former seems to be the more reasonable.

On account of its uncertainty, I have said nothing with reference to the diagnosis of an exudate due to a neoplasm. Quincke, however, claimed that one is able to diagnose carcinoma cells, in that they are

larger, more numerous and more polymorphous than endothelial cells. The nuclei of the carcinoma cells are much larger in proportion to the cell body than is the case with endothelial cells. We must not forget, however, that the endothelioma is not an uncommon tumor of the pleural cavity.

In regard to the actual practical value of cyto-diagnosis, as is the case with most new discoveries in medicine, in the beginning, too much was expected from this means of diagnosis, and, naturally, it has required time and study to settle this point. We must admit that in practically no case is the diagnosis in regard to etiology to be made from the cell-content of the fluid alone. The character of the cell exudate depends not alone upon the etiology, but also, at least at times, upon the severity of the infection. The presence in fluid of lymphocytes to the exclusion of other cells is one ear-mark of tuberculosis. The presence of polymorphonuclear leucocytes, in a percentage of 75 or more, points very strongly in favor of its being caused by an acute non-tuberculous inflammation. The occurrence of endothelial cells in large number, especially if they are in sheets, is one indication of a transudate, such as may be found in heart or kidney lesions.

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EMPYEMA.*

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As employed in the subject under consideration, the term empyema refers to the accumulation of pus in the pleural cavity. Professor Holt, in his work on diseases of children, describes the primary process as "an inflammation with the production of serum, fibrin and pus, at first accumulating in small pockets. The adhesions finally break down, the whole cavity becoming filled with pus, or with septa of adhesions remaining, the pus may become sacculated." Fibrinous exudates may cover both parietal and visceral pleura and, by extension, may involve the outer surface of the pericardium. The pleura is thickened and upon removal or absorption of the pus, becomes adherent and remains so after the recovery of the patient. Effusions within the pleural cavity may be primarily purulent or at first serous, becoming purulent later in the course of the disease. In a case of double pneumonia, with a left-sided effusion and pericarditis, which occurred in my hospital service, I drew off a quart of serum, at the end of the second week of the disease, which upon centrifugation, showed a considerable number of leucocytes. A few days later a quart of purulent fluid was removed and, with the insertion of two large-sized drainage tubes, the patient made a good recovery. A considerable quantity of fluid, a flocculent exudate, or a moderate amount of pus, may be disposed of by nature without surgical interference, but this is not the rule as regards the latter condition. Saccula-

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tion, due to partial adhesions, may so modify the physical signs as to cause doubt in regard to diagnosis and embarrassment in locating the pus during operation.

The cause of purulent exudates in the pleural cavity is an infection from micro-organism, such as the streptococcus, following, in the majority of cases, an acute pleurisy; the pneumococcus, accompanying a pneumonia; a double infection associated with a tuberculosis; a metastasis as occurs in puerperal infection, typhoid fever, or, in fact, the introduction, into the pleural cavity, of any pus-forming microbes through traumatism, resulting in injury to the pleural membrane, such as stab wounds, gunshot wounds or fracture of the ribs. The etiology can be made out only by a careful study of the clinical history in each case.

The direct microscopic examination of the exudate for diagnostic purposes, where tuberculosis is suspected, can not be relied upon, as an endless amount of labor might be entailed without detection of the tubercular bacillus. Cultures should be made and examined and guinea-pigs should be inoculated where circumstances will permit, in order to remove doubt as to the presence of tuberculosis. Tubercular cases are rarely pure infections. They are usually mixed with streptococcus, staphylococcus or pneumococcus. A tuberculous exudate in the pleural cavity may follow an acute dry pleurisy, serofibrinous pleurisy, or it may develop without any premonitory symptoms. The insiduous manner of its appearance may be illustrated by the following case, seen in consultation: The attending physician suspected fluid in the pleural cavity. The patient, a man of 40, was a teamster and, until a few days previously, had followed his vocation. His heart was displaced to the right by a left-sided exudate. His only complaint was difficulty in breathing and, when told of his condition, was disposed to doubt it, as he had suffered so slightly from the accumulation. About half a gallon of bloody serum was withdrawn, which soon reaccumulated, and, with fever and loss of strength, the patient died a few weeks later, after a second removal of the fluid.

In my service at the Springfield Hospital during the past two years I have treated a number of cases of empyema arising from pneumonia, one from a general infection following a miscarriage, one from a gunshot wound of the lung and in consultation I have also seen one resulting from a stab with a knife and another from a bayonet. The latter presented nothing unusual, but the case following the miscarriage may prove interesting.

This was a case of a woman of 25 years of age, having one son of about 4 years of age. She miscarried at three months and developed fever shortly afterward, followed by general infection resulting in multiple abscesses in different parts of her body, including the lower extremities and the arms. Eight abscesses had been previously opened, when she came under my care at the hospital with a discharging sinus in the sixth intercostal space on the right side, at the site of an incision which had been made by her former attending physician. This was inadequate for emptying the pus, so I resected a rib in order to provide for more

complete drainage. The pus in this case was due to a streptococcus infection. She left the hospital and returned to her home in a neighboring city before the cavity had healed, and I do not know the subsequent history of her case.

Within the past two years I have seen, in consultation, four cases of metastatic purulent accumulation in the right pleural cavity, two of them following attacks of appendicitis and one an abscess at the right of the ascending colon, while the fourth came into the hospital with a diagnosis of an inflammatory process in the gall bladder. I made an autopsy on the case of perityphlitic abscess and found multiple abscesses throughout the liver and another at the base of the lung, which was doubtless the focus from which the empyema began. All of the other three cases were operated upon, but the patients died from general infection. I have seen, in my personal experience in general practice, one case where a serous exudate was removed from a patient, a young married woman, who had made a rather tedious recovery from a continuous fever of indefinite history. She came to the city and consulted me in regard to a cough and a painful condition in her right side. The cause of the symptoms proved to be fluid in the pleural cavity. In the course of the blood examination on account of the previous history, Dr. Shuts made a Widal test with positive reaction. A quart of fluid was drawn from the pleural cavity, in which typhoid bacilli were demonstrated.

While these cases do not all come strictly within the scope of this paper, they are referred to in order to emphasize the fact that the source of infection may be remote, having their origin as seen in the appendix, intestinal tract or the gall bladder.

The diagnosis of an exudate in the pleural cavity is, as a rule, not difficult when a systematic and thorough physical examination is made. The conditions with which it is most frequently confounded are pneumonia, pulmonary tuberculosis and tumors of the mediastinum and pleura. The diminished or absent respiratory murmur, diminished vocal fremitus and voice transmission, frequently enlargement of the affected side, with occasionally bulging of the intercostal spaces, flatness upon percussion, with a sense of resistance on the affected side, skodaic resonance, marked tympany at the apex in many cases, the so-called William's tracheal tone, obliteration of Traube's semilunar space in the left-sided exudates, especially in adults, and displacements of the heart, all taken together, point to exudates in the pleural cavity, in contradistinction to the other conditions mentioned above.

From my personal observation I am inclined to attribute the most frequent source of error to a misconception of the position assumed by the exudate. I have frequently observed physicians trying to establish a horizontal line for the exudate with the patient in the sitting posture, under the impression that the fluid will follow the force of gravity, while, as a matter of fact, this is the exception rather than the rule. The dulness is usually higher posteriorly, unless the amount of pus is quite large, owing possibly to the continued reclining position assumed by the patient and the tendency of the compressed lung to recede in the

direction of the least resistance. The much-discussed "S"-shaped line of Ellis, if present at all, is so variable from day to day as to be of minor importance. Nor can the character of the fluid be always determined with certainty. Chronicity, with chills and variable temperature, point rather to purulent exudates, although I have seen patients convalescing from an attack of pneumonia with none of these symptoms, and get up and carrying with them three pints of pus in the pleural cavity. It was my privilege, at one time, to listen to an able diagnostician devote two lecture hours on two consecutive days to demonstrating, by fine points of discrimination, why a pleural exudate in the patient before us must be serous rather than purulent. He reserved the puncture until the last in order to demonstrate carefully the signs of differentiation. Upon introducing the aspirating needle, he was surprised and not a little chagrined to obtain a thick, fetid pus instead of the serum that the symptoms and physical signs seemed to indicate. It should also be borne in mind that bronchial breathing and even a marked vesicular murmur may both be transmitted through considerable accumulations of fluid in the pleural cavity. Twice during the past winter have these phenomena been demonstrated in cases under my care; once in a case of empyema following pneumonia, from which a large quantity of pus was removed, and again, in a case of double chylothorax, where, with the assistance of Dr. Patton, I removed a half gallon of chylous exudate from the pleural cavity. While the careful clinician will attempt exactness in the interpretation of all symptoms and physical signs, these facts only emphasize the advisability of resorting to the comparatively harmless aspirating needle for the purpose of removing every element of doubt as to diagnosis.

In regard to the treatment, there can be but one course to pursue from the time the diagnosis is established, and that is the prompt removal of the exudate and thorough drainage of the pleural cavity. The long and tedious delay in recovery, in the cases which have fallen under my personal observation, has, almost without exception, been attributable to inaccurate interpretation of the character of the fluid and a consequent resort to doubtful and inutile methods to promote its absorption. With the means at hand, an exploration can be made with so little discomfort and danger to the patient that all doubt of the diagnosis should be promptly removed by an early introduction of the aspirating needle, in all cases where that procedure is indicated by the physical signs. An occasional failure to confirm the diagnosis in this manner, where the signs are not so pronounced, while it does not deter me from pursuing the same course again under similar conditions, has taught me to state frankly to the patient and family that the aspirating needle is to remove all doubt and is only preliminary to a more radical operation in case the diagnosis is confirmed. In a case where the physical signs seemed to leave no doubt as to the diagnosis, I saw a very eminent diagnostician make eight intercostal punctures before the suspected exudate was obtained. The possibility of error should not cause us to hesitate in employing promptly every means of establishing an early diagnosis, rather than subject our

patient to not only the continued suffering, but to the danger of the increasing accumulation and certain prolongation of convalescence.

It would appear unnecessary to emphasize this simple and comparatively harmless method of exploration, were it not for the fact that I have seen patients up and about, after an attack of pneumonia, where, upon examination, the signs pointed unmistakably to the presence of an exudate in the pleural cavity. These were not always in cases where the attending physician failed to recognize the condition, but where, owing to failure to obtain pus upon the first attempt, further exploration was abandoned, either from apprehension upon the part of the patient or diffidence on the side of the operator. In one case of six months' standing, following pneumonia, I introduced the needle in three different places with negative results, only to be called again a few days later to find my patient greatly relieved by the discharge of a large amount of pus through the bronchus. This patient had spent several months in Colorado in an attempt to recover from a supposed tuberculosis, but finally made a good recovery after Nature had come to the rescue. Again let me emphasize prompt action in order to remove every element of doubt as to diagnosis. When the existence of pus is established, free drainage is imperative. A number of methods of procedure are at our disposal and must be selected according to the history of the case and the conditions present. I shall only briefly refer to one or two embarrassing conditions that may arise after operating upon these cases.

In one of the cases referred to above, the patient was attacked with severe coughing and a rather alarming syncope, both of which would probably have been avoided had I withdrawn the fluid less rapidly. In operating before adhesions have taken place or the lung had become, in a measure, fixed, the entrance of air into the pleural cavity may cause labored breathing and, in one case of a right-sided pleurisy, in a young man of 17 years, on the morning following the introduction of the drainage tubes, I found the heart displaced still farther to the left than before the operation and an apparently almost complete collapse of the lung with increased dyspnea. This condition gradually disappeared and the patient made a good recovery. The negative pressure devices of Perthes and Van Hook for syphon drainage are certainly ingenious and, in two instances, I have regretted not having resorted to their use. These instruments have been described by their authors in the medical journals, and I will not consume your time in discussing their very apparent merits. In all later operations at least, resection of a rib at a point most favorable for drainage has the almost unanimous approval of the profession. A few years ago it was my custom, in the older cases, to wash out the pleural cavity and introduce an emulsion of iodoform. A few operators used, at this time, saline solutions, one washing out after the operation, but if the lung is capable of expansion the vast majority of uncomplicated cases progress favorably under thorough drainage, without any other treatment.

As such a large number of empyemas follow pleurisy and pneumonia, I have almost entirely abandoned the cumbersome and exceedingly

questionable jackets, poultices and pastes used in the treatment of these diseases, in order to make daily examinations, so as to detect, at the earliest moment, any accumulation in the pleural cavity. I feel confident that this method, if persisted in by all of us, would materially reduce the mortality.

GENERAL SYMPTOMATOLOGY AND PHYSICAL SIGNS OF PLEURITIS.*

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Systematic discourses on pleuritis are accustomed to make a distinction in the symptomatology between the dry and exudative forms, pleuritis sicca, and pleuritis humida. While it is quite true that some generalized pleurisies run their entire course with little or no exudation of fluid, yet in the majority of cases they represent successive stages in the same process. In the average case of pleuritis the scene is opened by slight chilly sensations, usually repeated several times, but only in the severest cases attaining the dignity of a well-marked rigor. Following the chill, the temperature rises with moderate rapidity. While on the one hand rigors are uncommon, we have, on the other hand, a very considerable number of cases where the initial chilly sensations and subsequent rise of temperature are so slight as to make but little impression on the patient. We have all seen cases of pleuritis, with considerable fluid present, in which the subjective symptoms had been very slight, and the patient explained them by saying that he had caught a slight cold. In a case which recently came under my observation, a young woman in apparently perfect health was attacked by what her family physician took to be a simple cold. This lasted for nearly two months, without either fever, pain or dyspnea ever reaching such a point as to lead her to consider herself more than slightly ill. At this time a feeling of weakness led to another examination by her physician and the removal of five liters of fluid from her chest.

The expression of the face shows nothing characteristic, except a slight pallor or in a small number of cases cyanosis. Children may react, with high temperatures, to a very slight pleuritis, while, on the other hand, cases with diabetes, Bright's disease or carcinoma may have absolutely no rise of temperature. At the very beginning in a typical case there is a sharp, stabbing pain, beginning with the affected side and radiating to the back or around towards the median line in front. This stabbing pain is usually felt either during the entire inspiration or during one phase of it, and is, of course, greatly intensified by coughing. Cases are not so very rare where the pain is referred largely to the sound side. As Gerhardt has suggested, these cases are probably due to nerve anastomoses,

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although the latter have never been anatomically demonstrated. A point frequently overlooked in text-book discussion of pleuritis is the fact that a typical pleuritis may set in with no pain in the chest at all, but with well-marked abdominal pain, which may be referred to the gall-bladder or inguinal region. These abdominal cases of pleuritis are the exact counterpart of the abdominal cases of pneumonia, and so acute and sharply localized may be the abdominal pain that it has lead, in many instances, to the diagnosis of appendicitis, cholecystitis, etc. I know personally of one case where the abdomen was opened, and many more are recorded in the literature. I have elsewhere called attention to these cases, which only emphasize the correctness of Austin Flint's dictum that the examination of no case is complete without an examination of the thorax. The pain may assume one of two characters: first, and most commonly, of the pleuritic type proper, that is produced by the friction of the inflamed pleural surfaces against each other; second, it may be of a typical neuritic character and simulate closely an intercostal neuralgia.

The position of the patient is fairly characteristic. In the first stage the patient lies on the sound side. This rule has, however, many exceptions. The respirations show striking changes. They are considerably increased in frequency and of a short, staccato type. On the affected side the excursions of the chest are much smaller, and one may occasionally notice that the inspiration starts in later on the affected side. When the exudation becomes marked the patient lies on the affected side. Dyspnea may now become well marked, even to orthopnea, as, by sitting up in bed, the lung is, to a certain extent, unburdened of the weight of the fluid. Cough is an almost constant symptom and is of a particularly irritating variety, becoming much lessened when the leaves of the pleura are separated by fluid. Change in position is very apt to increase the cough. Expectoration is, in uncomplicated cases, practically absent. In the most severe cases a local edema may be noticed over the affected area of the chest, due to the direct propagation of the inflammatory process from pleura to subcutaneous tissue.

The temperature range in pleuritis is moderate. At the onset the temperature rises to 102° or 103° F. and remains at or about this height until the commencement of absorption, at which time it gradually declines. In a few cases, pleuritis acutissima, the temperature may rise to 105° F. These are cases which usually eventuate rapidly in empyema. The pulse range is moderate in frequency, rarely exceeding 100. In some cases it may be more rapid than would correspond to the degree of fever. The pulse tension shows only slight changes. Rosenbach remarks on the striking constancy of the blood pressure, even with exudates of considerable size. This is not necessarily a proof of normal conditions of circulation, inasmuch as with increased action of the vasomotors a diminished volume of blood in the arteries may be compensated. These considerations make it plain why it is that in a considerable number of cases of pleuritis, especially those with large exudates, we find symptoms of venous stasis. The pulse is of normal tension, but of small volume, and the venous system correspondingly overfilled. If cardiac insufficiency develops, symp-

toms of general venous congestion soon show themselves. We must bear in mind that circulation is not only impeded by pressure on the lungs, but also by the pressure of large exudates upon the heart and great vessels. The heart itself expands against higher external pressure than normal, and, all these taken together, make it very plain that the mechanical difficulties, which impede the circulation, are very considerable. The well-known fact that in large exudates a sudden active movement, such as sitting up in bed, may occasionally produce a fatal collapse is thus readily explicable. The pulsus paradoxus may be frequently observed, but has no especial significance nor importance. The urinary symptoms do not differ from those of any acute febrile process. The course of an acute serofibrinous pleuritis is not especially characteristic. In a mild case, at the end of a week, the temperature subsides and the effusions commence to be absorbed. With greater effusions, extending as high as the third or fourth rib, an average course would be from two to three weeks. In left-sided effusions the most reliable indication of commencing absorption is the increase in size of the Traube's space. The blood findings are simple and consist, in the majority of cases, in a mild leucocytosis, 10,000 or 15,000. In the tubercular cases this is wanting.

Physical Signs.—Inspection: In the first stage the affected side may be slightly but plainly drawn in as the result of the spasmodic contraction of the muscles on that side endeavoring to limit respiration of the affected side as far as possible. In the second stage the affected side is distinctly distended as a result of the increased intrathoracic pressure. The vertebral column may show slight curvature in the corresponding direction. If the effusion is of any considerable size, the displacement of the viscera may be plainly seen. The apex beat is no longer in its normal place, the liver is pushed down, and the epigastric pulsation may be seen to be greatly increased. Under favorable circumstances, in thin individuals, the lower end of the liver may be plainly visible, and in a left-sided effusion the left half of the liver may be pushed down until the lower margin is horizontal, or even below the level of the right lobe. In a right-sided pleurisy the reverse holds good, and the lower margin is sharply oblique from the right below upward and to the left.

Palpation.—The most characteristic phenomenon is the pleural rub, produced by the friction of the two inflamed surfaces. When very soft it can not be felt. It customarily disappears with the development of the effusion. It is ordinarily most plain at the margin of the effusion, and particularly in front and in the axilla, where the excursions of the lung are greatest. Pressure on the thoracic wall may increase it, as does also deep inspiration. The tactile fremitus is entirely absent over the lower portion of the effusion if this be only of moderate size. In children, however, quite fair-sized effusions may exist and the tactile fremitus be still present. In a typical case with considerable fluid present, the behavior of the tactile fremitus is characteristic. Below, where the fluid layer is thickest, it is absent, gradually increasing until it may be even intensified in strength at the upper margin, where the compressed lung conducts the

vibrations better. Some authors have stated that fluctuation may be obtained. I have never been able to make this out.

Percussion.—In the first stage this is negative. As soon as any considerable quantity of fluid is present dulness may be obtained, increasing in intensity to a wooden flatness below if the fluid layer be thick. Just below the clavicle, a Skoda's resonance, a peculiar, somewhat tympanitic note, is obtained, due to the relaxation of the lung tissue. The Wintrich's change, on opening the mouth, can not be elicited. It is often assumed that very small quantities of fluid give rise to the characteristic physical signs. Every one who has had much experience, and who has paid particular attention to this point, recognizes the fact that small quantities of fluid, 150 or 200 c.c., give rise to no physical signs. It is only when the amount of fluid exceeds 200 or 300 c.c., and then only under favorable conditions, e. g., thin thoracic walls, that dulness and the other characteristic signs can be elicited. In order to elicit absolute flatness, a fluid layer, having a thickness of from 5 to 7 centimeters, must exist at the spot percussed. Where flatness exists it is accompanied by a feeling of greatly increased resistance. The boundary line of the dulness is usually a curve, with the convexity upward. With light percussion and a medium-sized effusion, the line begins lowest behind, making an S-shaped curve upward toward the axilla, and then extending down in a straight line to the sternum. This applies only when the patient is in the erect position, Ellis' line. On the right side the flatness merges into the liver flatness. On the left side Traube's semi-lunar space may be diminished or obliterated. Occasionally, in medium-sized effusions, the mobility of the fluid may be brought out by first percussing in the erect and then in the recumbent posture. This is regarded as an infallible sign of fluid, but is by no means always to be elicited. Two other phenomena of percussion should be noted here, as they are highly interesting from a theoretical standpoint, although of almost no practical importance. They are: 1. bruit de pot fele. When the exudate is of considerable size, one can hear quite plainly, in the upper and anterior intercostal spaces, typical cracked pot resonance. This is due to the relaxation of the lung parenchyma. If this relaxation goes on still further, a dulness develops. 2. In very large exudates, where the lung is markedly compressed above the fluid level, percussion in the second intercostal space will give at times a markedly tympanitic note, the so-called Williams' tracheal tone. This phenomenon may at times be found in other conditions, such as pneumonia of the upper lobe, and is really nothing more than the percussion note of a large bronchus, obtained through an overlying consolidation. The Wintrich's change of tone may here be often obtained.

Mensuration.—Mensuration shows the increase in size of the affected side.

Auscultation.—The pathognomonic sign is the pleural friction rub, which can be heard in many cases where it can not be palpated. It may occur only in inspiration, or only in expiration, frequently during only a part of inspiration. If the pleurisy extends to the vicinity of the heart, this will, of course, be influenced by the contractions of that viscus, and

we have a pseudo-pericarditis, or a pleuro-pericarditic friction rub. Over the entire chest inspiration is apt to be jerky and somewhat diminished over the entire affected side. This initial enfeeblement of respiration is due to the pain attendant upon inspiration, and differs from the diminished breath sounds in the stage of effusion in that it is heard over the entire side of the chest. With the effusion, the breath sounds grow less and less and show the same characteristic behavior as the percussion note, i. e., the breath sounds are only slightly impaired above, growing less and less audible below as the layer of fluid grows thicker.

A most interesting phenomenon is the occurrence of distant bronchial breathing, when the fluid attains a considerable size. The compression of the lung, caused by the fluid, produces the necessary physical conditions for bronchial breathing, and this may be heard in some cases over the fluid, apparently coming from the depths of the chest, which, as a matter of fact, it does. In children the bronchial breathing may be so loud as to lead to the diagnosis of pneumonia. Over the sound lung the breath sounds are loud and harsh as the result of the dyspnea. On auscultation of the voice, which is particularly valuable, we find the corresponding signs. With even small effusions there are weakened voice sounds. Under those conditions, where we hear bronchial breathing, we may have a beautifully demonstrable bronchophony. This may occasionally take on a bleating quality, egophony. One would suppose, *a priori*, that the whispered voice would never be heard over an effusion. This is, however, not true. Bacelli has called attention to the fact that the whispered voice can be heard with great plainness, over quite large effusions, so long as these remain serous. In purulent effusions the whispered voice is absent, due probably to the enormous numbers of cellular elements in the fluid, which interfere with the sound propagation.

The Roentgen pictures are characteristic, but of small practical importance, except in the diagnosis of interlobar pleuritis.

With the absorption of the effusion, a leathery, creaking friction rub may again be heard when the leaves of the pleura come in contact with each other (redux-crepitus).

Pleuritis Interlobaris.—Occasionally fluid may accumulate between the upper and lower lobes of the lung. These cases are most puzzling. The physical signs are often obscure and consist in a band of dulness, corresponding to the line of separation of the two lobes. The majority of these cases are purulent, and rupture into a bronchus is not infrequent. These cases are rarely correctly interpreted without exploratory puncture.

Pleuritis Diaphragmatica.—These cases are rare, but I have seen a few exquisitely marked cases. The leading symptom is intense pain of a stabbing character, on breathing, with nothing in the physical examination to account for it. Considerable quantities of fluid must accumulate before the physical signs of effusion are manifest. Icterus is not infrequent in these cases, and has its origin in two facts: 1, the greatly diminished diaphragmatic action, which is an important accessory cause in the maintenance of the bile flow, and, 2, the extension of the inflammatory process to the liver.

Encapsulated Pleurisy.—In rare instances the effusion may be encapsulated in some atypical place, although by far the commonest condition is to have an encapsulated pleurisy remain as a residue of a considerable-sized effusion. The physical signs consist in a localized dulness, with the corresponding changes in the breath and voice sounds. A circumscribed friction rub may often be obtained. The cases are difficult of correct interpretation without exploratory aspiration. It is in the atypical cases, particularly the interlobar and encapsulated forms of pleurisy, that the Roentgen picture may be of considerable diagnostic value.

Resorption of the Exudate.—Pleuritic adhesions: Resorption of the exudate is characterized by a gradual return of the above-mentioned physical signs to the normal. This proceeds very slowly, and in particularly severe cases a very long time may elapse before the percussion note comes back to the normal. A complete *restitutio ad integrum* occurs only in the smaller effusions. After practically every large effusion some degree of thoracic deformity is left, due to the contraction and organization of the fibrinous exudate. This shrinking (Schrumpfung) of the exudate leads to a drawing in of the chest upon the affected side, particularly in the lower lateral portions of the thorax, and this phenomenon reaches its maximum in children or youths of delicate build. The circumference of the chest on the affected side may be diminished by some centimeters, the ribs are drawn closer together, the scapula is approximated to the vertebral column, the anterior portion of the chest is flattened, and, lastly, scoliosis develops. A moderate dulness and a diminution of vocal and tactile fremitus remains, due not to persistent fluid, but to the fact that the adhesions which remain are of sufficient thickness to produce these physical signs. On the opposite side a vicarious emphysema may develop. A very common result of this combination of pleural adhesions on one side with vicarious emphysema on the other is the production of hypertrophy of the right heart, with subsequent dilatation; cardiac insufficiency, and the long train of symptoms of venous congestion which follows in its wake.

PERICARDITIS; GENERAL SYMPTOMS AND PHYSICAL SIGNS; MECHANICAL EFFECTS OF FLUID DISTENTION OF THE SAC.*

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Little has been added to our knowledge of the manifestations of acute pericarditis since Stokes elaborated the significance of the pericardial friction murmur in 1831, but the manifestations of chronic adhesive pericarditis, on the other hand, are even yet ill defined and not well understood, and the diagnosis of chronic adhesive pericarditis is at the present time only occasionally to be made with any degree of positiveness.

Pericarditis is an important pathologic process and plays a much

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larger rôle in the etiology of heart diseases than is generally ascribed to it. There is always more or less myocarditis associated with pericarditis, and Broadbent goes so far as to state that many of the symptoms which are ordinarily ascribed to pericarditis are in reality due to myocarditis with cardiac dilatation.

Symptoms are, in the main, due either to the direct inflammatory involvement of the pericardium or of the contiguous structures, such as the myocardium, the pleura or the mediastinum, with involvement of the neighboring nerve trunks; or to pressure of a pericardial effusion; or to the results of a pericardial synechia. The varied and interesting clinical picture of chronic adhesive pericarditis, especially in those cases where the pericarditis is but one of the manifestations of a multiple serositis, has especially attracted the attention of clinicians during the past few years. Of the subjective manifestations of pericarditis pain is the most frequent, although pericarditis is more often painless than painful. This is in direct contrast to the painfulness of pleurisy. Pain is usually present in dry pericarditis, and is ascribed to the rubbing of the rough pericardial surfaces one upon the other. It may disappear in a few hours with the accumulation of effusion, but this not without exception.

In the more chronic forms of pericarditis pain may be due to the direct involvement of the nerve trunks in the inflammatory process, as, for instance, the vagus, the phrenic or the periaortic plexus. With effusion, pain may be due to the direct pressure of the distended sac upon the surrounding organs, such as the heart muscle, the lungs or esophagus. Pain may be due to secondary manifestations of adhesive pericarditis, as the pain of passive liver congestion. Pain may be retrosternal, precordial, epigastric or abdominal. It may be continual or paroxysmal, or may assume the type of an angina pectoris, with radiation to the shoulder, the trigeminus region or to the abdomen, simulating the pain of gastric ulcer, gallstones or peritonitis (Neusser, Ortner, Stokes, Gibson).

Neusser is of the opinion that coronary sclerosis does not entirely explain all of the phenomena of true angina pectoris, and he ascribes to pericarditis, with its involvement of the periaortic plexus, a rôle in the production of angina pectoris in many cases. In plexus angina of pericardial origin, as taught by Neusser, the radiation of the pain is without rule, and may be through any of the many communicating branches to the brachial or cervical plexices, or through the vagus to the abdominal region. The pain may be paradoxical, in that it may start at the hand and run up the arm and across the shoulders (Roseman).

Vagus involvement may be manifested by slow pulse, globus, larynx spasm, singultus, dysphagia or gastric phenomena, such as vomiting, belching or gastric pain (Neusser). Such vagus involvement may be noticed in some cases of acute dry pericarditis, as well as in pericarditis with effusion. Pain may be elicited, in some cases, by direct pressure over the course of the vagus and phrenic nerves in the neck, in the peristernal line or by pressure over the diaphragm.

Subjective manifestations, other than real pain, are often prominent, such as precordial uneasiness, oppression, or pressure, tightness, palpita-

tion or an ill-defined distress and anxiety in the chest. Hyperesthesia over the precordial and epigastric regions is common, and may be out of all proportion to the objective findings. The slightest irritation, even the pressure of light bed covering, may be intolerable. Dyspnea is a frequent symptom, and is variously ascribed to the accumulation of fluid interfering with cardiac and pulmonary activity, to myocarditis, to venous engorgement or to pressure on the vagus or arterial trunks. It is usually a prominent symptom, when an effusion accumulates rapidly, and may be absent even in cases of immense effusion of slow development.

Dysphagia is a rather uncommon symptom, and is ascribed to direct pressure upon the esophagus. Loss of voice may be due to paralysis of either the right (Eichhorst) or the left (Gibson, Sibson, Stokes, etc.) recurrent laryngeal nerves. Fever is not at all distinctive of pericarditis, and while often present, it assumes the type that is characteristic of the particular form of infection present. Fever may precede the friction rub by a few days in rheumatic pericarditis, especially when the fibrin deposit begins posteriorly. Fever may rise with the formation of a large effusion, but this is not without exception.

On inspection, the manifestations noticed are those due to either effusion or chronic adhesive pericarditis. In effusion, one may notice a precordial bulging, especially in children; the left chest may lag on inspiration; the first rib sign of Ewart may be present, and the intercostal spaces may be widened. The apex beat may be present or absent. When present it may be inside the area of absolute dulness; when absent, it may often be made to reappear when the patient leans forward in a sitting position. Systolic retraction, at the apex or on the left lower posterior axillary region (Broadbent), may be present, but is in no way pathognomonic of either concretio- or acretio-cordis. This sign may be simulated in cases of hypertrophied heart, in which the increase of negative intrathoracic pressure at systole causes a retraction of the intercostal spaces.

Ascites appearing with a minimum or no edema of the lower extremities—the so-called *ascites præcox* (Neusser)—speaks especially for a pericardial obstruction to the return venous circulation. This form of ascites is, however, only suggestive of chronic adhesive pericarditis, and is in no way peculiar to it. This was beautifully illustrated by a case that went to postmortem recently in Wesley Hospital. The patient, a young man, had gone to Mercy, Cook County, or Wesley Hospitals some twenty odd times, suffering from passive congestion of the liver and ascites of maximum degree. Edema of the legs was minimal or only moderate, until the terminal attacks, when it became prominent. The heart was negative except for a relative mitral systolic murmur, which would disappear each time after a few days' rest in the hospital. Each time the ascites and dilated liver would disappear rapidly under rest and profuse catharsis, so that the patient would return home after a few days' hospital care in fairly good condition. The case was studied by many clinicians at the various hospitals, where the patient was a familiar figure, and the concensus of opinion was that the condition was produced by some obstruction to the return venous circulation, mechanical in

nature, probably pericardial. The patient had a terminal left-sided hemiplegia and left-sided hydrothorax. The postmortem showed myocarditis and cardiac dilatation of maximum degree, the pericardium and hepatic veins were normal throughout. This case was of especial interest, in that it illustrates the difficulty one encounters in making a definite diagnosis of chronic pericarditis in some cases, and that such diagnoses should only be made with reserve.

On palpation, a pericardial friction fremitus may be demonstrated, in cases where a friction rub may be heard. It gives one the impression of being superficial, and has no definite focus of intensity. It varies much as to seat and extent, and is, in general, of the same significance as the pericardial friction murmur. The pulse of pericarditis is not destructive; it may be empty and compressible where cardiac activity is interfered with. A pulsus paradoxicus is said to indicate pericardial synechia, but this manifestation occurs in so many other conditions, as well as in health, that it is of little diagnostic value.

On percussion, in cases of fluid accumulation, the area of absolute dulness is characteristic. With small amounts of fluid, of 100 c.c. or over, the dulness appears at the base of the heart, the right liver-heart angle becomes obtuse (Ebstein), and the right cardiac edge loses its normal convex outline, as may be demonstrated upon careful percussion.

With greater accumulations of fluid, the well-known triangular area of dulness, with base directed downward and blunted apex upward, is usually present. That this is not always true of large accumulations of fluid, is emphasized by Thayer, in an interesting article published recently in the Johns Hopkins Hospital Bulletin (May, 1904), in which the dulness appeared in the left side of the thorax in an atypical manner, and also by a case described by Ortner, out of Neusser's clinic, in which the accumulation of fluid was mostly posterior. The area of cardiac dulness may usually be increased by having the patient lean forward, and an area of dulness may sometimes appear posteriorly, at the angle of the scapula, as described by Bamberger.

On percussion one is often able to demonstrate a movable line of lung resonance, upon deep inspiration, about the borders of cardiac dulness, a point which speaks against acretio-cordis.

Since the discovery of the pericardial friction murmur by Collins, in 1824, the friction rub has generally been considered the most characteristic manifestation of pericarditis. It is not absolutely pathognomonic of pericarditis, yet, when it is present, in its typical form, it usually means pericarditis. It is variously described as rubbing, purring, churning, musical, metallic, creaking, harsh, soft, etc., but these adjectives can not in any way convey an accurate impression of its characters. Its peculiar acoustic properties can not be described. It is so characteristic that when it is present in its typical form it is rarely to be confused with any other clinical manifestation. The one thing which is more characteristic of it than any other quality is its variability. It is variable as to its time of appearance and disappearance, variable as to location and quality of sound. The area over which it is best heard varies, not only from day

to day, but, in some cases, from hour to hour, and occasionally the rub may be present but a few hours altogether. It may vary in rhythm. It may be a simple to-and-fro murmur one day, and a triple murmur the next, and, on the other hand, its rhythm may vary with the successive heart beats under the stethoscope, with or without change of position of the body. The pericardial murmur seldom corresponds exactly, as to rhythm, with the sounds of the cardiac cycle. It is more apt to occur in late systole, or late diastole, or may be interposed between systole and diastole. This is due to the fact that the pericardial friction is at its maximum at the time of maximum expulsion of blood, which is late in systole, and it is also due to the fact that the rhythm of the pericardial friction murmur depends to a great extent upon the accidental position of the deposit of fibrin.

It may be single, double or multiple. A single friction rub is usually systolic, but it may, in rare instances, be diastolic (Ortner). A double murmur is usually presystolic-systolic. A triple murmur may be presystolic-systolic-diastolic. Multiple murmurs may be due to lateral motion of the heart, or to a split ventricular systole. The pericardial friction rub is described as being "superficial" and "near-to-the-ear." It is not usually propagated to any great distance from its point of production, as is so characteristic of endocardial murmurs. Pressure of the stethoscope often increases its intensity. It is influenced by gravitation, and may be heard only when the patient assumes certain positions, or may be intensified when the patient leans forward in the sitting position. It is modified by respiration, usually being intensified at the height of inspiration, but occasionally at the height of expiration. It is increased when intrathoracic pressure is increased, as in the well-known Valsalva experiment of taking a long breath, closing the glottis and exerting pressure by contracting the abdominal muscles, thus increasing the pressure of the pericardial surfaces one on the other, favoring the production of a pericardial rub.

As to location, the pericardial friction rub is more often heard at the base of the heart, over the sternum, in the third or fourth left intercostal spaces near the sternum, or in the tricuspid region. It may, in rare cases, be heard only at the apex, especially late in the course of the affection. This is due to the fact that the apex, being the most movable part of the cardiac surface, may be the only part which has escaped pericardial fixation. In the early stages of pericarditis, the friction murmurs are never heard beyond the cardiac region. While pericardial friction murmurs may be heard in more than one spot, yet they are almost always confined to the cardiac area, and are not to be heard in the back. There is no definite focus of intensity (Sibson), as is so often present in endocardial murmurs, yet they are usually distinct over the whole audible area.

The common belief that when the exudate appears the friction rub disappears is erroneous, in that a friction rub at the base may often be present even with immense accumulations of fluid. The usual teaching of the German school, following the lead of Skoda, which is that on

account of the fact that the heart has a greater specific gravity than that of the surrounding fluid, the heart gravitates to the posterior part of the sac, in cases of large effusion, is erroneous, at least in some cases, as is conclusively shown by Thayer in the article previously quoted. The relation of the heart to the chest wall is not materially changed by the accumulation of large amounts of fluid, it being held in about the normal position by the great vessels at the base. One of Thayer's cases, with an accumulation of 1,200 c.c. of fluid, still showed the heart in contact with the anterior chest wall. This being true, the presence of a friction rub does not, therefore, preclude the presence of a great accumulation of fluid.

On auscultation about the periphery of cardiac dulness, in cases of effusion, one may hear crepitant râles on inspiration, which are the expression of atelectasis at the compressed lung edge, about the dilated pericardial sac. This finding may, in some cases, be used to outline the extent of pericardial distension, especially where the precordial dulness is maked by emphysema (Neusser).

DIAGNOSIS OF EFFUSIVE PLEURISY AND PERICARDITIS.*

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The recognition of effusions in the pleural and pericardial cavities may be very easy or exceedingly difficult, but it is always important. In this paper no attempt will be made to cover every phase of the subject, in order that as much time as practicable may be bestowed upon those features which are of the greatest importance. The large majority of the cases of pleurisy with effusion are tubercular or pneumonic, although the other varieties should not be forgotten. In the tubercular series of cases, the pleurisy may be the initial symptom; it may occur early, or before much destruction of pulmonary tissue has occurred; it may appear as cavities approach the pleural surface, or as a terminal feature. In the first form, the patient is usually suddenly seized with an acute pain in the side of the chest, which is increased by coughing or deep inspiration. The pain, after a few hours, gradually subsides, is noticeably lessened in acuteness at the end of 24 hours, is markedly better at the end of 48 hours and has nearly or quite disappeared by the end of the third day. There is, usually, a repressed cough, which may be so slight as to escape ordinary notice; it may be absent. Expectoration is not a symptom. Moderate or slight fever is present during the first few days, and may persist for a week or more. After a few days dyspnea upon exertion may appear, slight in most cases, moderate in some, severe in a few and absent in some others. This symptomatology has direct bearing upon the diagnosis, inasmuch as all such cases should be subjected to such careful physical examination as will reveal pleuritic effusion if present.

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The pleural friction rub will have been sought for and probably detected, which confirms the fact of a pleurisy. If the affected side be examined in a good light, it will be noted that the respiratory excursions are noticeably impaired, from slight retardation to nearly total immobility, due to the induced pain or slight effusion in the first instance, and to a distending quantity of liquid in the second. There will be no bulging of the intercostal spaces, unless there is a very large effusion, one which inhibits diaphragmatic movements upon the affected side. If early, deep pressure is made in the intercostal spaces, pain is induced; by such palpation the surface area of the affected pleura may be outlined. If effusion is present in any considerable quantity, there is dulness on percussion. In outlining the area of dulness, light or moderate percussion should be employed; heavy strokes bring out modified resonance from any permeable lung which may protrude into the liquid. In moderate effusions, the upper border, as outlined by percussion, is an irregularly curved one; it changes slowly and slightly upon change of position. In massive effusions, the note is not materially altered by varying the force of the stroke; the upper line of dulness is straighter; the diaphragm is motionless and depressed, with consequent depression of the liver, if the right side is involved, and displacement of the heart to the right, if the left side is affected; the intercostal spaces may bulge. Respiratory and vocal fremitus are more or less impaired in proportion to the extent of the effusion; in massive ones, they are absent, except in children, in whom they may persist.

In the presence of these symptoms and signs, it is reasonably certain that a serous effusion is present, but this should be verified by puncture by a hollow needle attached to an aspirating apparatus, which, for purely diagnostic purposes, may be that of a large hypodermic syringe. The strictest antiseptic precautions should be taken to prevent additional infection. The puncture should be made where dulness is most marked, and at the greatest distances from resonant areas, provided, always, that it is made sufficiently high to avoid the diaphragm. The seventh or eighth interspace, a little in front of the angle of the scapula, is a favorite and appropriate location. Sufficient liquid should be removed for microscopic and other diagnostic examinations.

In pneumonic pleurisy with effusion, the diagnostic difficulties are notably increased. The liquid poured out may be small or moderate in quantity and lying immediately in juxtaposition to solidified lung, with the result that a diagnosis by percussion, and possibly by auscultation, may be impossible. This may be especially true in effusions at the base. In these cases, the liquid may appear early, and this probably is oftener the case than is generally supposed. The fibrous deposits upon the visceral and parietal pleura, with its strong and early tendency to form close adhesions, has an inhibiting influence upon effusions at a late period of the attack. So far as I know there are, with one exception, no symptoms indicating the occurrence of effusion; it must, therefore, be detected by physical signs. The pain follows the course common to all cases, with or without effusion; it gradually diminishes in acuteness and

severity and usually disappears within three days. With extended involvement of lung and pleura, the pain persists or recurs. The cough and expectoration pursues the ordinary course, as does also the temperature range. It is rare—I have never seen it—for an effusion in connection with pneumonia to be so large, early in the disease, as to cause bulging of the intercostal spaces and depression of the liver. In a single instance, I have seen the heart displaced toward the right with consolidation of the upper lobe of the left lung and with effusion at the base, with compression of the lower lobe. In this case, frequent observations revealed the rapidly gradual accumulation of liquid and the disappearance of vesicular breathing, some bronchial breathing remaining audible in the back. In this case, the flaky serous effusion of the fourth day became the lightly purulent one of the day following. Such cases must be rare. The exception to the rule, that symptoms are not significant, lies at the end of the attack; the fever does not subside at the usual time, or, if it does, it speedily reappears. Under these circumstances, the condition is often called unresolved pneumonia, or relapsing pneumonia. The symptom is so significant, however, that no such case should be left with such a diagnosis, without a most painstaking exploration of the pleural cavity. In the vast majority of instances, pus will be found. In these cases, the effusion is not infrequently sacculated, either between the visceral and parietal pleura, or interlobar, and, in either case, the difficulties of diagnosis are greatly enhanced.

The physical signs are similar to those found in tubercular cases. The friction rub may be oftener obscured; the respiratory excursions are earlier and more noticeably impaired. The area of involvement may be accurately outlined by intercostal pressure. Dulness on percussion may be due, wholly or in unmeasurable degree, to the pulmonary consolidation. It has been asserted that the sensation given to the finger in pulmonary consolidation is that of greater resistance than in simple effusion, and I have thought that such were the sensations conveyed to my finger, but, with more prolonged experience, I am not sure that much stress should be placed upon the fact, if fact it be. The limiting line of dulness seldom follows that of tubercular effusion, due probably to the disturbing factors of character of liquid present, the more or less numerous adhesions formed, the extent and location of the pulmonary consolidation, etc. Auscultation discloses diminished breathing or absence of respiration, but here, again, are to be encountered the disturbing factors of pulmonary consolidation, as may be readily understood. It follows, therefore, that in pneumonic pleurisy with effusion we must rely for diagnosis mainly upon exploratory aspiration. In every case of pneumonia in which there may be detected symptoms or signs of pleuritic effusion, recourse should be had, promptly, to this measure. In the vast majority of cases, the period at which the injury becomes pressingly pertinent is during the second week of the attack. In the presence of symptoms such as above mentioned, the diagnostic aspirating needle should be used intelligently and painstakingly until the presence of liquid in the pleural cavity, free or sacculated, has been proven or tentatively dis-

proven. It is important to note that if liquid is not obtained, even after many punctures, the evidence is not conclusive that it is not present. The heavy and viscid character of the effusion may, rarely, be the obstacle, even when the cavity containing the liquid has been penetrated. The most frequent difficulty lies in the failure of the needle to penetrate the thick false membranes which line the parietal pleura, or, these becoming detached, may be pushed in front of the needle. In either instance, failure may result, or more careful technic may surmount the impediment. Although in pneumonic effusion, as in tubercular cases, the point where the liquid is most likely to be encountered is in the seventh or eighth intercostal space, posteriorly, near the angle of the scapula, yet the rule has so many exceptions that this location should be selected solely because here the dulness on percussion and absence of breath sounds may be most profound. If liquid is not found at the base, in the presence of indicative signs, the interlobar spaces should be very thoroughly explored. In one of my cases, the area of most pronounced dulness was found high up in the axilla, and here, at a great depth, pus was discovered. In my opinion, there are no serious objections to making multiple punctures, but, in order to lessen the danger of infection from the surface, several pleural punctures may be made from one made in the skin. The needle should have a large bore, and the most scrupulous antiseptic precautions should be taken. Microscopical and cultural examinations of the removed liquid will give valuable information.

Pericarditis with effusion occurs especially in connection with rheumatism, scarlatina, pneumonia and tuberculosis. In rheumatism and tuberculosis, the effusion is nearly always serous. In scarlatina, it is also usually serous, but in some of the severest cases the liquid is thin, sero-purulent and very virulent. In pneumonia, a small, sero-fibrinous effusion is not rare. Diagnosis in these may be difficult or impossible, but, because of the natural history of these cases, this question does not often arise. The large and obtrusive pericardial effusions in pneumonia are usually purulent, although they may be sero-fibrinous. In any of the morbid conditions mentioned the pericardium should be subjected to frequent and thorough investigation, in order that early and accurate information may be had of the presence of such effusions, although those of large and dangerous character are not very common.

Passing over the symptoms, and such physical signs as may be obtained from inspection, palpation, etc., as affording information of but little value, and none which can not be obtained from other and more reliable sources, we come at once to those elicited by percussion and auscultation. In approaching this subject we should be careful to note the facts, regardless of theoretical considerations, which are, or may be, misleading. Note, for example, that the effusion distends the lower portion of the pericardium, notwithstanding the fact that it is of lower specific gravity than the heart.

By means of careful, light percussion it is possible to outline, with accuracy, the area of direct contact of the heart against the chest wall, and by heavier strokes to obtain dulness over that portion of the heart

which is covered by lung tissue. The outlines shown by these areas are well known and require no description. In this connection it is well to bear in mind that the apex beat of the heart corresponds to the lowest and extreme sinister aspect of the heart. Now, in pericarditis with effusion in detectable amount, the cardiac area of dulness is enlarged; at the lower portion it broadens out in direct proportion to the quantity of liquid present, giving rise to three of the cardinal findings of this condition, namely, the dulness is carried to the left distinctly beyond the apex beat of the heart, the area of dulness upon deep and superficial percussion are identical, because the thin tongue of pulmonary tissue which normally overlaps the left border of the heart is pushed to the side, and the abolition of the normal resonance in the cardio-hepatic angle at the right side of the sternum, a sign to which Rotch has called particular attention. With large effusions the borders of dulness are everywhere enlarged, exaggerated below, but very marked above also. The outline which is now presented is distinctly indicative of pericardial effusion, inasmuch as the appearance is unlike any other condition.

Auscultation reveals the apex beat within and above the extreme left and lower margins of dulness, and in addition the cardiac sounds are feeble and appear to be distantly located. The respiratory sounds do not lap over the line of dulness, even on deep breathing. These are important diagnostic features. In some cases of large effusions there may be heard, near the angle of the scapula, or somewhat anteriorly to this point, after the patient has been for a time in the supine position, distinct bronchial breathing; if he leans forward or occupies the prone position the bronchial breathing gradually gives way to crepitant râles. The bronchial breathing is accompanied by noticeable dulness, which changes to a more or less resonant tone on percussion with the appearance of crepitation. In some of these cases and in some others of large effusions there is to be found dulness on percussion between the angle of the scapula and the spine, as pointed out by Ewart.

The physical signs enumerated are found only when the pericardium is non-adherent. Should adhesions be present, either as the result of previous inflammation or during earlier stages of the current attack, the effusion may occupy surface cavities of greater or lesser extent and variously located. Such accumulations of liquid cause asymmetrical bulgings which may be difficult or impossible of diagnosis.

Puncture of the pericardium for diagnostic purposes is usually undertaken only in those cases with large effusions in which the diagnosis, from other signs, is already probable. It is, therefore, a confirmatory measure, ordinarily preparatory to operative treatment. The needle should be introduced in the left fifth intercostal space, as near as possible to the sternum. In very large effusions the puncture may be made in the corresponding space on the right side, or on the left side near the extreme border of dulness, outside the apex beat. The object, in any instance, being to enter the pericardial sac without wounding the heart.

It is to be regretted that the limits of this paper will not permit a fuller consideration of the subjects which have been passed in short re-

view, and that many important matters are entirely ignored. It must be said, however, that in this important field expertness in diagnosis can only be attained by careful observations made upon cases personally investigated.

TREATMENT OF PLEURISIES AND PERICARDITIS.*

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Infection plays a part in causing all inflammations of the pleura and pericardium. Unfortunately, we possess no means of specifically combating most infections and especially those which commonly affect these serous membranes. Therefore, radical or specific treatment is impossible. We must endeavor to ameliorate distress, to avert dangers and to treat symptoms. Inflammations of these structures are clinically classified as acute fibrinous, acute with serous effusion and chronic. The methods of treatment relied upon to-day are different for each of these groups of cases.

The prominent symptoms of acute fibrinous inflammation are pain, tenderness, cough and fever. Each of these or any two of them may be insignificant or wanting. Cough does not accompany pericarditis, unless the pleura and lungs are simultaneously involved. It may be an inconspicuous symptom even in pleurisies. In many cases fever is also slight or absent. Pain is the most constant and most striking symptom. Often it is of very moderate severity and needs no treatment or is felt keenly only when a deep breath is taken or when a patient coughs or sneezes. In such cases, applying adhesive strips on the affected side, so that deep inspiration is impossible, will ameliorate discomfort. Strapping, however, is not sufficient in the severer cases and must be supplemented or substituted for by other means. Heat, especially the moist heat of a poultice, is very comforting. But the frequent change of dressings needed to keep poultices hot is often a trouble and annoyance.

An ice bag is equally effective in pericarditis and, after the first feeling of coldness has been forgotten because the skin is benumbed, is not uncomfortable. It has to be renewed less frequently than the poultice. It is also much easier to maintain steadily a degree of cold than of heat. But, owing to the size of the thorax and its shape, it is impossible to apply a bag of ice so that it will stay in place and cover the thorax. It is also too heavy. However, rubber coils, through which water is kept flowing, can be used advantageously. As these coils are not commonly at hand, they are much less frequently used than poultices. In pericarditis I prefer a bag of ice to a poultice. It must not be forgotten that heat and cold produce other effects besides lessening pain, although that is the first noticed and is most striking. In pericarditis a bag of ice slows the heart, lessens fever when it is present and is believed to combat the vascular and

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nutritional changes which are a part of inflammation. Both hot and cold applications have a similar antiphlogistic effect.

The proprietary clay pastes are used very frequently. I believe them to be of doubtful utility, because, although they are applied hot and retain heat fairly well, they are not changed as often as they should be. Often they are allowed to remain for 12 or 24 hours upon the chest. It goes without saying that the paste is then and has been for many hours at the temperature of the body and has not been, in reality, a hot application. I have not been able to convince myself that they deplete, as is claimed, because of the hydroscopic property of the glycerin which they contain. A clay paste makes a useful poultice, but it must be changed frequently if the effect of a hot application is to be had. In passing, let me call attention to the pharmacopeal preparation called cataplasma kaolini, which any pharmacist can make and which is as good as any of the proprietary pastes and cheaper. Sinapisms will quickly relieve mild grades of pain and, because of the ease with which they can be prepared, are commonly employed. The more active cantharidal preparations are little better and are certainly not to be preferred to poultices or a bag of ice in these cases.

Blood letting, for such inflammations as pleurisy and pericarditis, is not resorted to in these days. But many treatises on this subject still commend the application of leaches to the chest. However, this practice, although efficient, has also fallen into disuse. In a few rare cases pain is so intense that morphin must be given by mouth or beneath the skin. It must be remembered that whenever morphin is used to relieve pain it should be given efficiently, but not longer and in doses no larger than is needed. When pericarditis is due to rheumatism, the salicylates should be used. It is rare, however, that pleurisy is caused by it or requires anti-rheumatic treatment. Opiates are needed oftener to lessen cough than to lessen constant pain.

When there is much fever, a feeling of well-being is acquired most quickly by promoting elimination from the kidneys and bowels. Ammonium acetate and potassium acetate and citrate are useful to prompt the kidneys and magnesium citrate, sulphate or sodium phosphate are good purges. The temperature is rarely so high or so constant as to make cold baths a necessity, but there is no objection to them when it is high. Bags of ice or cold water circulating through rubber tubes coiled upon the chest will often lessen bodily temperature as well as produce the antiphlogistic effects already alluded to and will make cold baths unnecessary. Chemical antipyretics are harmful if used day after day in such cases, for they lessen the oxygen-carrying power of the blood, increase cyanosis and interfere with normal metabolism.

When we have to do with acute inflammation with serous effusion, the treatment just outlined is the best during the first ten days, except in the rare cases in which the effusion is poured out with great rapidity and, therefore, suddenly compresses a lung or the heart. It is well in dry pleurisy and pericarditis to prescribe a dry diet in order to prevent the development of an effusion; and, when an effusion is demonstrable,

the same diet is required to prevent it from becoming great or to hasten its absorption. By a dry diet is meant a minimum amount of proteid and starch-bearing food, and, if possible, not more than two glasses of fluid daily. The latter should be taken occasionally in sips. The food must be restricted in amount and in kind, so that the fluid contained in it will not be great. Very little of fruits and other foods especially rich in water should be permitted. Such a diet as this is especially useful, and I might cite many cases illustrating its good effects.

However, much more will be accomplished if, at the same time that a patient is confined to a dry diet, elimination of fluids from the body is promoted as vigorously as possible. I have found nothing so uniformly useful for this purpose as purgation by elaterium. Clutterbuck's preparation is the best. It can be given in doses of grm. .008 to grm. .015 once or twice daily. Very rarely do I find patients made uncomfortable by it. Salines, especially magnesium sulphate, is oftener used, although I have not found it as efficient or generally as agreeable to patients. When salines are given they should be dissolved in as small a quantity of water as possible and should be given in doses sufficient to produce copious liquid stools. Diuresis and sometimes diaphoresis is resorted to as an aid in bringing about elimination, but neither is as efficacious as catharsis. Such diuretics as potassium citrate and acetate are the best and they should be given in full doses.

When there is a liquid effusion, pain is not felt, for the congested and often raw pleural or pericardial surfaces are separated. Nevertheless, blistering the skin over the affected part is useful, first, as an antiphlogistic measure and, second, to aid elimination. Cantharidal collodion or blistering plaster should be placed over the tenderest points, if any such can be found. These applications should be accompanied or immediately followed by fomentations, in order to promote the filling of the blisters and afterward the continued abstraction of serum from the surface thus made raw. This will accomplish best the second object of vesication, namely, the abstraction of fluid. From my own trials of this method of treatment I have been led to conclude that too little fluid is thus drawn off to be of much consequence. However, if there is tenderness over the affected part of the thorax or other evidence of persistent and considerable inflammation, the blisters do lessen it. Therefore, when there are such indications, I feel that blistering is desirable.

Many physicians apply externally tincture of iodin instead of cantharidal preparations. I have no confidence in its efficacy. It does not act so promptly nor so vigorously.

The iodid of potassium or sodium is of little use when the exudate is liquid, but when it is thick, fibrinous and cellular these drugs are beneficial, because they promote the more rapid absorption of such exudates. Under the same circumstances, calomel or other preparations of mercury given in moderate doses, repeated 3 or 4 times daily, are beneficial, for, as is generally conceded by clinicians, they stimulate the absorption of such exudates and lessen the liability of their being organized so as to make permanent, thick pleural or pericardial walls or adhesions.

When, in spite of treatment such as I have outlined, a pleural cavity or the pericardium is distended steadily more and more with fluid or after two weeks' duration an effusion is not absorbed, we are compelled to think of other means for its removal. Paracentesis is a safe, prompt and efficient one. If dyspnea is considerable and cyanosis is evident, paracentesis should be resorted to at any time; also whenever positive pressure is produced by the effusion, so that the heart is displaced and a lung greatly compressed. The fluid should be withdrawn by an aspirator or by trocar and cannula. If the effusion, even though it is considerable, causes no displacement of the heart and little dyspnea, paracentesis may be delayed for two or three weeks or sometimes even longer. However, if there is some persistent fever, and especially if the chest wall remains tender, it is not best to delay the withdrawal of the fluid more than two weeks, for under these circumstances there is usually a persistent pleural inflammation, which may lead to so great thickening of the visceral pleura as to prevent the perfect re-expansion of the affected lung. This deformity is less likely to be produced if the lung is permitted to expand before the pleura has grown too thick.

If, however, there is no fever and there are no tender spots between the ribs, and especially if the pleurisy is suspected to be tubercular, longer delay is permissible and even desirable, because tuberculous lesions, for the time being quiescent, are more apt to be kindled into fresh activity when the lung is allowed at once to re-expand and to become functionally active. Tuberculous pleurisy should be treated as other forms of pulmonary tuberculosis are by keeping the patient quiet, out of doors and by feeding as generously as the effusion permits. Treatment out of doors in a dry and moderately elevated climate will promote quickest recovery from the tuberculosis and quickest absorption of exudate. It will most certainly prevent the patient reinfecting himself with tuberculosis, which he is likely to do if he is treated indoors, in rooms imperfectly ventilated. It will also most certainly prevent infection by pus organisms, which are so apt to complicate such cases and hasten destructive changes in the lungs. If the fluid in either thoracic cavity is pus, it should be removed not only promptly but also permanently by free drainage. This treatment is surgical and you will not expect me, in the present discussion, to describe it in detail.

We must be guided, in aspirating the pericardial cavity, by very similar rules. It should be done when the cavity fills with rapidity and produces cyanosis and pericardial distress. If these symptoms do not exist, it may be long delayed, although the effusion is great. However, when the pulse is weak, irregular or very rapid, or is become so, even if effusion has taken place so gradually as not to have caused much distress or cyanosis, aspiration is indicated. It is my rule, both in pleurisy and pericarditis, if symptoms are such as make it desirable to determine whether the fluid effusion is purulent or not by withdrawing some of it, to do so in such a way that as much of the fluid as possible can be abstracted at the time. For, if the patient must feel the discomfort of

the needle thrust into his side, it is well to use the opportunity to effect therapeutic results as well as diagnostic. The technique of paracentesis is so well known that I need not describe it anew.

Permanent pericardial adhesions can not be removed by treatment. They may be so slight as to cause no appreciable trouble or the bands binding the pericardial surfaces ultimately may be stretched so as not to interfere with the function of the heart. But they sometimes lead to a degree of cardiac hypertrophy and at last to cardiac weakness or even fatal asystole. Rest or graduated exercise, cardiac and general tonics are then needed to prolong life. The problem is one of treating the heart-muscle rather than the pericardium.

Retraction of the chest-wall, which follows pleurisy not infrequently, often can be prevented or corrected by a prompt removal of the patient to a dry climate in a mountainous country, where perfect and rapid absorption of exudate will be effected and fullest expansion of the lungs assured. So soon as convalescence is established, gentle walking exercise should be encouraged and, later, carefully graded hill climbing. Such treatment can be carried on safely only under the constant supervision of a physician. Various substitutes for this treatment have been tried. The pneumatic cabinet or other contrivance to effect pneumatic differentiation (as the inhalation of a relatively compressed air and exhalation into a rarified one is technically called) can be employed with benefit. However such devices are rarely at hand.

When retraction of part of the chest-wall has been produced, if it is not of too long standing, help can be obtained by blowing exercises, such as playing a horn and by arm exercises which insure the fullest chest expansion as well as by climatic change and hill climbing. In the most chronic cases, some compensating emphysema and even hypertrophy of the lungs may be produced by such treatment and it will insure also greater endurance, less breathlessness and better general health. A gymnastic drill, which is useful in such cases, consists of the usual calisthenic movements of the arms, legs, and body, but each movement should be repeated from four to six times in quick succession with the lungs as fully expanded as possible and held so during the exercise. After four to six movements made thus, a rest of a few seconds should be taken, before the same or other movements are made. It has been suggested that by bending the body toward the unaffected side, the affected lung would be more perfectly expanded. It has seemed to me that no better results and usually not as good ones were obtained in this way as by using both sides simultaneously, and as far as possible symmetrically. In addition to the drill just mentioned, an exercise of value is to direct a patient to grasp rings or a bar above his head and to practice lifting his weight. This exercise will help to stretch a side which is deformed, and will be made more efficient if the patient is sufficiently expert to fill his lungs fully while thus suspended. However, to accomplish much in any of these cases of deformity, perseverance and a long time are required.

PATHOGENESIS OF ASCITES.

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Ascites is an accumulation of free fluid in the peritoneal cavity. It may be the result of an inflammatory process in the peritoneal cavity, or may be due to some local or general disturbance not accompanied by inflammation. I shall consider only the non-inflammatory variety, as the inflammatory type is the subject of another paper. The non-inflammatory variety may be considered under two great headings, i. e.: Ascites as a part of a general dropsy; ascites as a purely local dropsy, caused by some disease in the peritoneal cavity.

Ascites as a part of a general dropsy occurs in a great variety of conditions, but especially in cardio-vascular, renal and pulmonary diseases; in diseases of the blood and the various forms of cachexia. In these conditions it is a secondary factor of a general process, and it will be necessary to consider, briefly, the pathologic processes involved in its production. In a general way, all cases may be classed under four heads: 1, Ascites due to stagnation of the blood in the blood vessels; 2, ascites due to interference with the absorption of the lymph; 3, ascites due to a disturbance of the capillary secretion—alteration in the capillary walls; 4, ascites due to some alteration in the chemical composition of the blood and lymph—a disturbance of their molecular concentration.

One or more of these factors are at work in every case of ascites, and in some cases all four may be present. In chronic valvular heart diseases, with broken compensation, we have stagnation of the blood in the venous radicles and capillaries; a change in its chemical composition due to deficient oxygenation; and, as a result of this deficiency, changes occur in the capillary walls as well.

The cells lining the capillary walls must be considered as living organs, endowed with a capacity for work, i. e., a specific secretory function. Any disturbance of nutrition results in a disturbance of function, so that the secreting cells lose their specific selective action and the capillaries become inactive tubes, allowing filtration to take place indiscriminately. The crystalloids of the blood pass through into the lymph, resulting in a lymph rich in chlorids and sugar, but relatively poor in albumin molecules. This condition favors further abstraction of fluids from the blood, thus increasing transudation, until edema or dropsy results. If this process occurs beneath the skin, we call it anasarca; if in the pleural cavities, hydrothorax; if in the pericardial cavity, hydropericardium; if in the peritoneal cavity, we call it ascites. Reuss has shown that the lymph, the transudates in edema, in pleural and pericardial effusions, in cerebrospinal fluid and in ascites are all deficient in albumin molecules, but rich in crystalloids, as compared with the blood.

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REUSS' TABLE.

Pleural effusion (transudates)	22.5 per M.
Pericardial effusion (transudates)	18.3 per M.
Peritoneal effusion (ascites)	11.1 per M.
Edema (subcutaneous)	5.8 per M.
Cerebrospinal fluid	1.4 per M.

Ketley and von Torday, Widal, Hamburger, Heidenhain and others have shown that there is an accumulation of chlorid molecules in the transudates and body tissues, greater than exists in the circulating blood. I was able to confirm these results, both by a chemical determination of the chlorids and by estimation of the freezing point. I found that ascitic fluid was always more concentrated than the blood of the same individual. When I say more concentrated, I mean that it contained more chlorid molecules, as shown by a reduction in the freezing point. In ten cases of ascites in organic heart disease, the freezing point was never above —0.58° C., and the average for ten cases was —0.60° C. The blood, taken from the veins of the forearm at the same time, froze at —0.56° C., which is normal. Here, again, we have two solutions separated by a thin membrane; one, the more concentrated ascitic fluid, in the free peritoneal cavity, the other, less concentrated, in the capillaries. The result is self-evident, namely, that, in an effort to establish an equilibrium, the more concentrated ascitic fluid will abstract more fluid from the capillaries, thus increasing transudation. The same is true of pleural and pericardial effusions, but to a lesser degree. Widal and his assistants have shown that the law of osmosis is quite an important factor in the production of subcutaneous edema. Heidenhain believes that the specific function of the capillary cells plays a controlling part in the formation of lymph, and whenever the removal of the lymph is interfered with and fails to keep pace with its production, edema results. He was able to increase the production of lymph remarkably, by subcutaneous injections of crab infusions, extract of leeches and mussels, or peptone solutions. In his experiments on dogs, he was able to increase the amount of lymph passing through the thoracic duct from six to fifteenfold. From these results he concludes that those substances are true lymphagogues and act by stimulating the secreting cells of the capillary walls. It is very probable that irritation of the vasomotor nerves also results in an increased vascular secretion, thus becoming a factor in the production of edema or ascites.

In renal diseases we have changes in the composition of the blood, brought about by a retention of waste products in the circulating blood, which act as vasomotor irritants, thus increasing vascular secretion. The direct action of these toxins on the cells of the capillaries causes a disturbance of function and allows filtration to take place. The changes in the cardiovascular system, associated with nephritis, cause increased blood pressure in the arterial system and favor transudation. The continued loss of albumin causes a state of hydremia, which favors the production of edema. If we add to all of these factors the effect of diminished excretion by the kidneys, the process becomes still more complicated.

In pulmonary diseases, as emphysema and fibrosis, we have deficient oxygenation of the blood, cyanosis, cardiovascular disturbances and subsequent changes in the capillary walls, rendering these more permeable to fluids.

In the blood diseases, as pernicious and secondary anemias, the leukemias, Hodgkin's disease, splenic anemia and Banti's disease, we have the chemical changes in the blood, the hydremia, the changes in the capillary walls, and, in addition, a toxic element to deal with. This also applies to the various forms of cachexias, such as that due to carcinoma, tuberculosis, syphilis and diabetes. Meltzner thinks that, in certain cases, we have a condition which he has termed "poto-cytosis," meaning an unusual capacity for fluids on the part of some of the tissue cells, thus favoring transudation.

Before considering the next variety of ascites it may be well to recapitulate and state briefly the factors entering into the production of edema or ascites. They are: 1, Venous stagnation; 2, increased permeability of the vessel walls; 3, hydremia or serous plethora; 4, increased secretion of the capillary cells; 5, changes in the composition of the blood; 6, interference with absorption of lymph; 7, vasomotor irritation by toxic substances increasing secretion of lymph and transudation; 8, high intravenous pressure as compared to relatively low arterial pressure; 9, retention of body fluids in nephritis; 10, potocytosis (Meltzner)—cell thirst.

Ascites as a strictly localized dropsy, caused by some disease in the peritoneal cavity. Under this head would come the different forms of cirrhosis, the atrophic, hypertrophic, biliary or Hanot's and the mixed form of cirrhosis; syphilitic disease of the liver, as gumma, interstitial hepatitis and capsular cirrhosis; carcinoma, abscess, cysts and amyloid disease of the liver; malignant tumors of the stomach and pancreas; tumors of the gall bladder and peritoneum; retroperitoneal and mesenteric tumors; sarcoma, tuberculosis of the lymphatic glands and peritoneum; aneurism of aorta, peritoneal adhesions, hypernephroma, thrombosis of the portal vein or its branches; ovarian tumors, as cystic adenoma, carcinoma of the uterus and urinary bladder, etc.

As a factor in the production of ascites, cirrhosis of the liver is of considerable importance. Rolleston states that ascites occurs in 50 per cent. of all patients dying with cirrhotic livers. It occurs in a vast majority of the cases of cirrhosis, which run their full course; that is, in cases not dying from some intercurrent infection or hemorrhage. In Prices' 142 cases, ascites was present at autopsy 72 times. Kelynack found it in 66 of 121 cases. The autopsy records at St. George's Hospital show that it was present in 84 of the 166 cases. In 80 cases, dying from the direct effects of cirrhosis, ascites occurred in 68 cases, or 85 per cent. It occurs with far greater frequency in the small than in the large cirrhotic livers. This is probably due to two factors: (1), because in the early stages of cirrhosis the liver is often larger than later in the disease, (2), because when cirrhosis becomes latent from compensatory hyperplasia of the liver cells the organ is considerably increased in size (Rolleston).

The factors which give rise to peritoneal effusion in cirrhosis require

some discussion. They are, I, mechanical factors: (a) obstruction to the passage of blood through the liver due to fibrosis and contraction around the intrahepatic branches of the portal veins; (b) thrombosis of the radicles of the portal vein in the liver; (c) thrombosis of the trunk of the portal vein. Obstruction to the passage of blood through the cirrhotic liver is an undoubted fact and leads to venous stasis in the portal vein area, as is shown by the collateral circulation and dilatation and thickening of the portal vein. That increased pressure in the portal vein alone does not always produce ascites has been shown by Hale White, who ligated the portal vein in dogs without producing ascites. Also. in patients with cirrhosis it is noticeable that when venous pressure is high, as shown by hematemesis and melena, ascites is frequently absent. Ascites is often present even though collateral circulation is well established, and, conversely, both ascites and all evidence of collateral circulation may be absent in cirrhosis. Levi reports such a case, in which, in addition, there was peritoneal tuberculosis, in itself a sufficient cause for ascites. For these reasons we may say that, although ascites is frequently present in cirrhosis, with obstruction to the passage of blood, this alone is not enough to produce ascites in all cases.

(b) Thrombosis of the radicles of the portal vein has been suggested as a factor in the production of ascites, but there is no positive proof for this assertion.

(c) Thrombosis of the trunk of the portal vein may and does produce ascites, but need not necessarily do so, and is an exceedingly rare condition, occurring only ten times in 334 cases of cirrhosis examined after death, or 3.3 per cent. of cases.

II. Toxemia plays a very important part in the production of ascites in cirrhosis. As a result of hepatic inadequacy, poisons absorbed from the alimentary tract and those produced in other parts of the body are not stopped and destroyed by the liver, but pass into the general circulation, and a condition of hepatic toxemia results analogous to that of renal toxemia. In both of these conditions, the patients become drowsy and frequently hemorrhages occur in different parts of the body. This hepatic toxin may have lymphagogue properties, thus becoming a more active agency in the production of transudates. The absence of ascites in biliary cirrhosis, where we have every evidence of toxemia, may be explained by the fact that in this type of cirrhosis there is less destruction of hepatic tissue than in the atrophic form, and, again, the toxins may be of a different nature. In biliary cirrhosis we have less interference with the passage of blood through the liver than in the portal type.

III. Ascites due to concomitant inflammation of the peritoneum is not an infrequent occurrence in cirrhosis, as we often find a perihepatitis, a low grade of inflammatory peritonitis, either the simple or the tubercular type. In these cases, we have an additional factor at work, because the hypertrophic inflammation of the peritoneum will interfere with the absorption of lymph, thus favoring the accumulation of free fluid in the peritoneal cavity.

IV. The influence of the cirrhotic processes on the cardio-vascular

system is a factor which can not be overlooked in the production of ascites. The backward pressure occurring in an ordinary case of heart incompetency would not be sufficient in itself to cause ascites, but, when associated with a cirrhotic liver it may readily do so. Heart disease may, then, produce ascites in patients suffering from cirrhosis in two ways, by backward pressure, as in tricuspid insufficiency, and by bringing about conditions, such as venous engorgement, stagnation of toxins, or damage to the endothelial cells of the peritoneum, which will precipitate ascites, due to the cirrhosis.

Summing up, we may again quote Rolleston, that ascites may be merely associated with cirrhosis and directly due to chronic or subacute peritonitis, perihepatitis or to cardiac failure. In rare cases it may be due to a direct complication of cirrhosis, as thrombosis of the portal vein or its radicles, or may be due to cirrhosis alone, as explained by the lymphagogue action of the hepatic toxins.

Syphilitic or capsular cirrhosis, gumma, carcinoma, ecchinococcus cysts, abscesses and amyloid disease would all act in a similar manner, with slight modifications in the pathologic processes. Tumors of the stomach, pancreas, mesentery and the peritoneum would act locally both by their pressure effects and as irritants. Malignant tumors would act also indirectly through their disturbance of the general metabolism, producing the hydremia of cachexia, intoxication and changes in the vessel walls. In these cases, especially in carcinoma of the peritoneum and tuberculous peritonitis, we have a peculiar type of ascites, namely, *chylous ascites*. This form is rare in cirrhosis, but we do sometimes have a so-called chylous fluid, which is very similar to it in appearance. Chylous ascites is found associated with carcinoma or tuberculosis of the peritoneum or mesentery; in infection with the filaria sanguinis hominis; compression of the thoracic duct by glands, scars or tumors; ulceration or traumatic injury to the lacteals in the mesentery; with thrombosis of the subclavian vein and with laceration of the thoracic duct.

Fatty or *adipose* ascites occurs in chronic and tuberculous peritonitis, in some cases of cirrhosis and is caused by the fatty degeneration of the endothelial cells and white blood corpuscles, which are in excess in the ascitic fluid.

Jousset has described a form of ascites which he calls the *opalescent*, because the fluid is not chylous or fatty, but is opalescent because lecithin and nucleo-albumins are present. *Hemorrhagic* ascites is sometimes found in cirrhosis, but more frequently in malignant diseases and blood conditions. There is still another form of ascites which is not well understood, namely, *fetal ascites*. Cases of this kind have been reported by Eden, Ballantyne, Spencer and Maline. The conditions found at autopsy were as follows: One case of portal obstruction, two cases of congenital syphilis, two cases of tuberculosis of the peritoneum and one case of hypernephroma of left kidney. I was able to find two cases in our autopsy records. In one case it was due to congenital syphilis; in the other, no cause could be found, except that the mother had died from eclampsia.

There were no pathologic changes in any of the fetal organs to account for the ascites.

With a view of determining the frequency of ascites and ascertaining, if possible, the factors at work in its production, I studied carefully the autopsy records of 3,000 cases at Rush Medical College and the Cook County Hospital, being careful not to count duplicates where such existed. Out of this large number I was able to collect 362 cases of ascites, which shows that it occurred in 8.3 per cent. of all cases coming to autopsy, or, in other words, in one out of every twelve bodies examined. In collecting these cases I took only those in which there was enough fluid present to warrant an anatomic diagnosis of ascites. An analysis of these cases shows that the ascites was a part of a general dropsy in 177 cases, or 49 per cent. of the whole. It was associated with cirrhosis of the liver in 77 cases, but cirrhosis of the liver alone was present in only 16. In other words, diseases of the heart and kidneys were associated conditions in 61 of the 77 cases of cirrhosis. Ascites was associated with disease of the heart alone in 27 cases; with some disease of the kidney alone, in only 14 cases; with diseases of the heart and liver in 21 cases; with diseases of the heart and kidney in 70 cases; with diseases of the kidney and liver in 33 cases; with diseases of the heart, kidney and liver in 38 cases. Ascites associated with some disease in the peritoneal cavity occurred under the following conditions: with carcinoma of the liver, stomach, pancreas, gall bladder, peritoneum or intestines, 49 cases; sarcoma occurred in only 8 cases; syphilitic gumma of liver in 18 cases; aneurism of abdominal aorta in 2 cases; tuberculosis of the peritoneum in 25 cases; chronic peritonitis, not tuberculous, in 4 cases; perihepatitis and capsular cirrhosis, 8 cases; endothelioma of peritoneum, 1 case; hypernephroma, 4 cases. Ascites was associated with, or occurred in constitutional and blood diseases as follows: in pernicious anemia ten times; splenic anemia two times; Banti's disease two times; Hodgkin's disease four times; splenomedullary leukemia four times; lymphatic leukemia three times; diabetes mellitus once; congenital syphilis four times; cachexia due to carcinoma, tuberculosis or malaria, twelve times; as a part of a general polyserositis, twice. In fetuses, ascites occurred in 2 cases, one congenital syphilis and one in a case of eclampsia in the mother.

A brief summary of the character of the ascitic fluid may not be out of place. In 277 cases the fluid was clear, usually of a light straw or yellow color, but sometimes more of a greenish hue. In many of these cases it was distinctly bile stained. In 72 cases the fluid was bloody. In four cases it was chylous or fatty. The fluid was bloody in carcinoma 33 times; in tuberculous peritonitis, 14 times; in organic heart disease and cirrhosis, 14 times, and once, each, in sarcoma, leukemia, splenomegaly and aneurism of aorta; twice in hypernephroma and syphilitic cirrhosis. Chylous fluid was found twice, associated with carcinoma, and twice in chronic ulcerative tuberculosis of the intestines.

From a study of these cases and what has been said above, it is very evident that the pathogenesis of ascites is not so simple as it would seem on first thought, but that many factors are involved in its production.

all of which must be thoroughly understood by the physician before a case can be treated successfully. This also shows the necessity of applying our physiologic and pathologic knowledge in the treatment of all diseases, and, most important, the necessity of a thorough and comprehensive examination of every case.

810 Warren Avenue.

SYMPTOMS AND PHYSICAL SIGNS OF PERITONITIS.

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CHICAGO.

Nothnagel describes two general groups of symptoms of peritonitis: (a) those which are the immediate result of inflammation and which may be called the "cardinal" symptoms, and (b) those which occur in infection, and which may be called the general symptoms. This is about as logical a classification as can be devised. Of the cardinal symptoms, pain, exudation, meteorism and abdominal rigidity are paramount; vomiting and hiccough, painful micturition and the general appearance of the patient being of second importance. Of the symptoms of infection, the blood changes, temperature and pulse demand consideration. The physical signs will be discussed with the symptomatology.

(A) Symptoms and signs which are the immediate result of inflammation of the peritoneum, the first four being of major and the last three of minor importance.

(1) Pain.—As a symptom this is of remarkable constancy. In acute cases it is always present, except in a few rare cases due to perforation, in whom death takes place before the peritoneum has had time to become inflamed. In chronic cases pain is present, if not spontaneously, at least upon pressure. No case is without it, even cases of tubercular peritonitis with an apparently latent course showing it, if carefully palpated. The severity of the pain varies in acute and chronic cases, acute peritonitis being one of the most painful of diseases because of the large amount of peritoneal surface involved. In most of the chronic cases the resulting adhesions, which greatly limit the surface inflamed, also greatly reduce the pain. The rapidity of onset in acute cases has a slight diagnostic value, those beginning very suddenly and violently being frequently caused by perforation from the alimentary canal or bladder, or from rupture of the spleen, as may happen in typhoid fever or malaria with very slight trauma. If the onset is more gradual, yet rather rapid, the peritonitis is due to some infection rather than to perforation.

The location of the pain is not in the skin, but in the peritoneum, as is shown by the fact that a fold of abdominal skin very carefully picked up by the fingers without disturbing the underlying peritoneum causes no pain, whereas very slight pressure downward upon the abdomi-

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* Read before the Section on Medicine, Illinois State Medical Society, at Springfield, May 15-17, 1906.

nal wall causes pain. In acute cases the abdomen is uniformly tender, but now and then a point may be found of greatest intensity, and this is apt to mark the site of invasion of the peritoneum. Often the pain is not confined to the abdomen, but radiates to the chest or shoulders. It will be remembered that the six lower intercostal nerves supply the anterior abdominal wall as well as the six lower intercostal spaces. Thus it happens that sensory impulses from the abdominal filaments may be interpreted by the sensorium as coming from the intercostal territory. Thus the pain is wrongly referred to the chest by the patient. Similarly the phrenic nerve, which is not wholly a motor nerve, contains numerous sensory filaments to the under surface of the diaphragm and to the uppermost sympathetic plexuses. Sensory impulses of pain from the upper portion of the abdominal cavity pressing upward through the phrenic nerve are thus wrongly referred to neck or shoulder by the patient or, indeed, to the anterior mediastinum. It is safe to say that pains referred to neck, chest or shoulder, therefore, indicate peritonitis, involving the upper part of the abdominal cavity, when peritonitis exists.

Very characteristic of peritoneal pain, whether in acute or chronic cases, is its exacerbation upon pressure. Abdominal pain which is eased by pressure is not localized in the peritoneum. Hence it is that in peritonitis the patient dislikes the pressure of bed clothes, and even the touch of the physician's hand. Spontaneous exacerbations of pain are due, as a rule, to peristalsis.

The effects of the pain upon the patient are numerous. In the acute and painful cases the patient lies with legs flexed on thighs and thighs partly flexed upon the abdomen so that the walls of the latter may be relieved and the underlying peritoneum escape pressure. The position of the trunk is such as to protect the abdomen. The patient can not move his legs without causing increased pain, but the pain that he has causes extreme restlessness and the arms and head are moved. Breathing becomes wholly costal to prevent movement of the diaphragm and is necessarily shallow. Thus cyanosis follows. Shallow breathing is rapid breathing, and hence the pulse rate is quickened. Coughing, sneezing, defecation and even talking are all suppressed so that the pain may be lessened.

(2) Exudation.—The exudate may be fluid or solid, free or encapsulated, much or little. The varieties are many: serous, fibrinous, purulent, hemorrhagic, sero-fibrinous, sero-purulent, chyliform. The exudate may even be gaseous when peritonitis follows perforation. A fairly large amount of fluid exudate must be present if it is to be recognized clinically, 200 c.c. in the case of a child, 1,000 c.c. in the case of an adult. Smaller amounts gravitate to the pelvis and escape detection, and on account of the pain from percussion a thorough examination is hard to make. The kind of the exudate can be determined only by making a test puncture. Its presence can be determined by two sets of signs, (1) the direct and (2) the indirect or secondary.

The direct signs of an exudate are the same as those for ascites and are based upon inspection, palpation showing fluctuation, and percussion. If the exudate is free in the abdominal cavity inspection will show a uni-

form distension in both flanks; if encapsulated, it will exhibit an asymmetry. For purposes of inspection it is best to have the patient in dorsal decubitus with the light admitted through one window in a slanting direction so that the shadows cast by any abdominal inequalities may show. This is a very valuable and much neglected method of physical examination. Fluctuation is not obtainable except when there is a large amount of exudate. Percussion is exceedingly valuable. If the patient is in the dorsal decubitus, an elliptical area of tympany should be present in the region of the navel. When the patient turns upon either flank this area of tympany should migrate to the opposite flank. A dulness which does not change position with change in the position of the patient is apt to indicate an encapsulated fluid. Auscultation is of slight value, except in rare instances of fibrinous peritonitis, where an exudate over liver or spleen gives rise to a friction rub which may be heard with the stethoscope upon inspiration, the rub being due to the fact that these organs are pressed down by the diaphragm in inspiration. At this point it may be added that collections of fluid within the intestines may be confused with exudates in the abdominal cavity. If splashing can be detected, air and gas are in contact, and, there being no signs of perforation, the fluid is within the bowel. Moreover, if there be no dulness over the symphysis pubis, the fluid is either within the intestines or else it is encapsulated within the abdominal cavity. If dulness in the two flanks be not symmetrical, a free exudate is not present.

The indirect or secondary effects of the exudate are exerted chiefly upon the diaphragm, heart, and circulation within the abdomen. The diaphragm is pushed high, well up to the fourth rib, thus compressing the lung and elevating the apex beat. The latter is not infrequently found in the fourth interspace. Since the diaphragm lies so high, its movements are much embarrassed and respiration suffers. Edema of the legs often appears because the exudate within the abdominal cavity ultimately reaches a tension great enough to compress the vena cava inferior sufficiently to interfere with the return of venous blood from the lower extremities. Hence the edema. The scanty urine is also explained by the great pressure of an ascitic exudate upon the renal veins. Such back pressure prevents proper excretion of urine.

(3) Meteorism.—The older authors used the word meteorism when gas accumulated acutely in the bowels, reserving the word tympanites for chronic cases; and it would be well for us to maintain this distinction. Meteorism and tympanites are intimately associated with intestinal paralysis, just as is the marked constipation of acute peritonitis. Yet not all cases show meteorism. It has been suggested that the paralysis of peristalsis is due to a nervous cause, as some alteration in the functions of the sympathetic nerve plexuses in the intestinal wall. Others account for the paralysis by edema of the intestinal wall. Ordinarily the more the gas the more the peristalsis, but it is not so in peritonitis. Complicating the explanation that meteorism is caused by paralysis of peristalsis is the fact that diarrhea occurred in many acute cases of puerperal peritonitis, and yet there is great meteorism.

As has been said, meteorism is not always present in the acute cases, and tympanites is very often lacking in the chronic ones. Though the symptom is a cardinal one, it yields in constancy to the pain and exudate. It varies exceedingly in amount, from an enormous distension to almost no distension at all, especially in chronic and encapsulated cases. If the patient's abdominal walls are flaccid the distension is naturally greater. Hence in puerperal cases meteorism may reach great proportions. The onset of meteorism is usually early, as a rule gradual, rarely suddenly. In perforation cases it comes especially early.

The effects produced by meteorism merely emphasize those produced by the exudate. The skin is smooth, shiny, tense and cyanotic all over the distended abdomen. The abdomen is apt to show more distension in the middle and upper parts in meteorism, with moderate bulging of the flanks; whereas a large exudate causes marked bulging in the flanks and less in the median and upper portions. The diaphragm is pushed high up, the apex of the heart often being displaced into the fourth interspace. The stiffer the walls the higher the displacement. Naturally respiration is greatly embarrassed, is shallow and rapid, and the skin is cyanotic in consequence.

The diagnosis of meteorism is, therefore, very easy. Inspection discloses its presence by the form of the abdomen. Palpation shows no fluctuation, thus differentiating exudation. Above all, percussion demonstrates the characteristic tympany. Auscultation may help; it is affirmed that if no gurgling be heard in the abdominal cavity for a period of five full minutes when the stethoscope is used intestinal peristalsis is present. Very important is the differentiation of meteorism, or gas in the bowels, from air or gas in the abdominal cavity following perforation. The old rule is the best we have: obliteration of liver and splenic dulness points to free gas from a perforation. I am aware that the intense meteorism of some cases greatly reduces liver dulness in the right nipple line, but it never completely eliminates it in the anterior or midaxillary line. If liver dulness is absent at these points it is safe to say that perforation has taken place.

(4) Abdominal rigidity is not present at all in many cases of chronic, circumscribed or adhesive forms of peritonitis; it is, on the other hand, habitually present in the acute cases and in not a few of the chronic forms. The rigidity is due to a reflex muscular spasm of the abdominal wall, and is probably caused by the sudden and violent irritation of the sensory nerves in the parietal layer of the peritoneum. Its purpose is clearly protective. Its onset is early, and, according to some authorities, it even antedates pain. As a rule, it does not persist, but has a tendency to disappear with the onset of intestinal paralysis. The effect of the abdominal rigidity is characteristic, since it precedes the exudate. The abdomen is flattened or even insunken, the edges of the ribs and the crests of the ilia being prominent. The wall feels hard and like a board. These signs are most marked in acute and perforative cases. In acute local peritonitis or focal peritonitis as appendicitis the rigidity is often

localized, one-half of the anterior abdominal wall being rigid and the other partially relaxed.

The symptoms and signs thus far mentioned, pain, exudation, meteorism and abdominal rigidity, are properly cardinal signs. The following three signs and symptoms, while very important, perhaps deserve secondary consideration. These are the general appearance of the patient, the vomiting and hiccough, and the scanty micturition.

(1) General Appearance of the Patient.—In the chronic cases there is nothing characteristic in the facial appearance. In the subacute cases emaciation and cachexia are common because of the anorexia. In acute cases there are many characteristic features. The tongue is dry and darkly coated; the breath is foul and cadaverous; the body is so disposed as to save all possible contraction of the abdominal muscles. Only the arms and head are moved restlessly about. The patient does not speak; coughing, sneezing and even breathing is suppressed. The facial expression is the so-called *facies Hippocratica*. The nose is pointed, every drop of fluid being taken from the lymph spaces and vessels, the eyes are sunken, the facial expression is anxious, the temples are hollow, the color of the face is lead-like or gray. Death looks from every feature. The extremities are cold and cyanotic. The vasomotor paralysis is complete. There is a cold perspiration on the forehead, about the upper lip, in the palms of the hand, along the calves, on the feet. The sensorium is markedly depressed, though the intellect may be preserved. If delirium is present it is of the low, muttering, bed-picking, typhoidal type. This is apt to be followed by loss of consciousness, stupor, coma and death from intense sepsis. If psychoses are present they are of the type of the infectious psychoses, depressive states predominating.

(2) Vomiting and Hiccough.—Vomiting is doubtless reflex, being due to irritation of sensory nerve filaments in the peritoneum. Hiccough is due to spasmodic contraction of the diaphragm caused by irritation of the sensory peritoneal filaments of the phrenic nerve with response in the motor supply. Hiccough is more common in acute than in chronic peritonitis because in the latter the area of peritoneum involved is in the pelvis or lower portion of the abdominal cavity. Sometimes the vomiting or the hiccough is excessively burdensome because of its frequency and because it persists a number of days. Vomiting is present in almost all cases with rapid onset, less frequently in cases with slow onset. In chronic cases vomiting and hiccough are unusual. Very important is the fact that vomiting almost invariably marks perforation.

(3) Micturition is painful and frequent and small in amount. This is because the peritoneum over the bladder is involved in general cases, and there is traction upon it as the bladder is emptied. Similarly the bladder does not fill because of the pain caused by even a partial stretching of the vesical peritoneum. The quantity is reduced both because of the fever and the small amount of fluid taken; and the specific gravity is high. Both nucleo-albumin and serum-albumin are present. Of the abnormal constituents, indican is the most notable. Normally present in very slight amounts, in peritonitis it is enormously increased, being ex-

ceeded only in cases of acute intestinal obstruction. Indicanuria in acute peritonitis is of marked diagnostic value; in chronic peritonitis, it has no diagnostic value whatever. According to Nothnagel, indican is a potassic indoxyl sulphate, derived from indol, which is a product of putrefaction of albumin, due to putrefacient bacteria.

(B) Symptoms and signs which are due to infection in general and, therefore, occur in infectious peritonitis.

(1) Blood Changes.—As a rule, the blood should be examined in a twofold way. The leucocyte count should be made and a blood culture started. Usually a leucocytosis is found, the average count being 15,000 to 50.000 white blood corpuscles per cubic millimeter. It is to be noted that the increase is almost wholly in the polymorphonuclear corpuscles, which rise from the normal, 69 to 70 per cent., to 80 to 85 to 90 per cent. There is no prognostic indication in the number of leucocytes found, but a leucopenia or a normal count are frequently found in the gravest cases. In these it seems as though the organism were too much overwhelmed by bacterial toxins to be able to react at all.

A blood culture frequently shows a bacteriemia. Hence endocarditis may complicate the case, though as a matter of fact it very rarely develops. Similarly pericarditis seldom occurs. Pleurisy is an occasional complication, infection being not so often by the blood as by the lymph channels of von Recklinghausen.

Temperature.—There is nothing typical about the fever. In chronic and circumscribed cases the temperature is low and usually little regarded by the patient. In acute cases the fever is high, as a rule, of sudden onset, often preceded by a chill. It is a continuous fever, reaching 104° frequently, but it may be very irregular with huge oscillations, especially when the case is approaching a fatal termination. Possibly there are three sorts of temperature worthy of special mention, (a) the temperature of perforation, (b) the temperature of sepsis, and (c) the temperature of tuberculosis.

(a) The temperature of perforation is a collapse temperature. If the temperature has been high, as in typhoid fever for example, when perforation takes place, it falls abruptly to the normal or below, while the pulse rapidly increases in rate, and the patient often vomits. The temperature usually remains low till death. (b) In sepsis the temperature shows great oscillations. It is usually accompanied by chills and a marked leucocytosis, and in such cases a localized collection of pus is to be suspected. (c) In tubercular peritonitis the temperature is low and insidious in early stages, reaching a maximum between 1 and 4 o'clock in the afternoon, frequently being normal a greater part of the night and forenoon. When diarrhea begins to appear the temperature rises and a continuous fever follows with no great remissions.

There are two facts associated with the temperature of great moment. The first is that such cases as show a difference of 2° to 4° between rectal and axillary or rectal and oral temperature are in a very bad way. In these cases the skin is cool, but internally the temperature is very high. The importance of taking the temperature by rectum in cases of

peritonitis is obvious The second fact is that there is no relation whatever between the degree of temperature and the character of the exudate. The abdomen may be full of pus and the temperature only 100. Indeed, one might almost declare that the temperature varies inversely with the severity of the case.

(3) Pulse.—In chronic forms there is nothing characteristic about the pulse. In acute forms it is so characteristic as to have received the name of pulsus abdominalis from the older authors. The rate is from 120 to 140, occasionally rising to 160 or 170. The tension is low, the pulse is small, thin and thready. It is not irregular, as a rule, but may be intermittent. In septic general peritonitis is to be found the highest pulse rate with the lowest tension in any infectious disease. Such a pulse is due to bacterial toxins absorbed in huge quantities by the vast surface of peritoneum inflamed. These toxins simply overwhelm the vasomotor and circulatory centers in the central nervous system. Thus the prognostic value of the pulse is very great, since it is to some extent an indication of the intensity of the toxemia. It must be declared, however, that there is no relation between pulse and temperature. Though they may go together in many diseases, in peritonitis each follows its own bent. The temperature may be slight or even subnormal and the pulse 160 or 170. Indeed, the higher the pulse rate and the lower the temperature the worse the prognosis. Moreover, there is no relation between the pulse rate and the character of the exudate. A purulent peritonitis may be associated with a pulse of only 100 or 110.

By way of summary, it may be said that the symptoms and signs of peritonitis fall into two groups: those depending upon peritoneal involvement and those due to infection in general. Of the former, pain, exudate, meteorism and abdominal rigidity are cardinal signs, the *facies Hippocratica,* vomiting and hiccough and meteorism being of almost equal importance. Of the symptoms due to infection in general, the blood changes, pulse and temperature are the most important. A leucopenia often denotes a grave case. Differences between rectal and axillary temperatures of 2° or over are ominous. The temperature is not influenced by the character of the exudate, and neither is the pulse. There is no relation between the amount of temperature and the pulse rate.

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THE DIFFERENTIAL DIAGNOSIS OF PERITONITIS AND OF ITS VARIOUS FORMS.*

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Peritonitis may arise in three ways: first, by direct extension from an infected focus, as from the gastrointestinal tract, from an infection of the gall bladder, etc.; second, by infection through the blood, as in pneumonia, sepsis, etc.; third, through the lymphatics, as from genital infec-

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tions, enteritis without perforation, etc. The chief bacterial causes are the colon bacillus, the pyogenic cocci, pneumococcus, tubercle bacillus and the gonococcus. The exudate may be fibrinous, serofibrinous, purulent, hemorrhagic, etc., or there may be only adhesions.

To consider first the diagnosis of acute general peritonitis, we find that it is based on certain symptoms and signs which may be briefly described in three groups: 1. Those due to the peritoneal inflammation itself. 2. Those due to functional or anatomic changes in the organs affected by the process. 3. The general symptoms.

Of the first group, the first is pain. This may come on suddenly if the peritonitis is due to perforation of the intestinal tract or of an abscess, or may come on gradually. As a rule, it is persistent, usually severe, increased by motion, and is located about the umbilicus. It may be absent where the patient is benumbed, as in typhoid perforation. It is usually associated with tenderness and rigidity. Another group of symptoms, due to the peritoneal involvement directly, is that due to an exudate in the peritoneal cavity. These vary with the amount and character of the exudate. There may be, for example, all the signs of free fluid; dulness in the flanks, shifting with position, fluctuation, etc.; but, as a rule, the fluid is less freely movable than in serous ascites and it may be so small in amount as to escape detection by ordinary methods. In this case it has been suggested to inject 500 c.c. of salt solution and remove it in hopes of finding signs of inflammation. Paracentesis gives the best means of diagnosis of a peritoneal exudate. Care must be taken that an adherent intestine is not punctured. The fluid removed will be turbid, of a higher specific gravity and show a higher albumin content than a transudate.

Of the symptoms, the effect of peritonitis on the abdominal organs, we may note: 1. Vomiting. This is fairly constant and persistent. The vomitus is frequently bile stained, but not, as a rule, feculent. It is reflex in origin. 2. Hiccough is frequent, even without diaphragmatic involvement. It is reflex in origin. 3. Constipation is the rule, although diarrhea occurs at times. The degree varies up to complete obstipation by paralytic ileus. 4. Meteorism is usually present. While due to intestinal paresis, it is, in a way, the cause of the latter or, at any rate, favors its increase by distention of the muscle coats of the intestine. It is often extreme. Paresis of the intestine may be diagnosed where intestinal gurgling can not be made out by the stethoscope. 5. Disturbances of the bladder, such as frequent and painful urination. 6. Indicanuria. This is the result of intestinal putrefaction and, according to Nothnagel, is of great diagnostic value.

The general symptoms of peritonitis are those due to inflammation and those due to collapse. The febrile reaction varies, but, as a rule, a moderate rise of temperature occurs and leucocytosis is usually found. Certain cases show, however, no rise of temperature and leucopenia. The collapse symptoms are rapid, thready pulse, drawn expression with hollow eyes—the so-called facies Hippocratica—cold hands and feet. As a rule, the pulse is a better guide to the patient's condition than the temperature.

The diagnosis of local peritonitis will depend on symptoms and physical signs that, on the whole, are much the same as in general peritonitis, but the clinical picture will vary considerably with the portion of the peritoneal cavity involved. In subphrenic abscess, for example, gastric symptoms and evidence of diaphragmatic irritation will be marked while in pelvic processes bladder irritation will be more frequent and the intestinal symptoms will be in abeyance. The local physical signs will be more in evidence and the history of some local process will be of importance in the diagnosis.

The diagnosis of acute general peritonitis, where the clinical picture is well developed, will, as a rule, be easy, but certain conditions will have to be excluded where the clinical picture is obscure. Intestinal obstruction, for example, may give rise to confusion by reason of its symptoms, especially nausea and vomiting, abdominal pain, obstipation and collapse. A rise of temperature may or may not occur. When it is remembered that peritonitis often develops as a result of the obstruction, the differentiation is seen to be even more difficult. The vomiting in obstruction, however, is more often feculent and the obstipation comes on earlier and is more extreme. The local findings will vary with the location of the obstruction. The intestinal distention may be local in case of obstruction high up, and a tumor mass may be present, especially in intussusception. Here, also, the age, the occurrence of bloody stools and tenesmus will assist in clearing up the diagnosis. Osler speaks of cases of peritonitis following appendicitis being diagnosed as intestinal obstruction. The frequency of the former condition should make this mistake rare.

Another condition that may give rise to difficulty in the differential diagnosis is acute hemorrhagic pancreatitis. The sudden onset with pain, nausea and vomiting, and fever with local signs in the epigastric region, usually lead to a diagnosis of intestinal obstruction or peritonitis, and, as a rule, it may be said that before operation the diagnosis can not be made. The condition has been suspected in several cases, however.

A third condition that may have to be differentiated is simple enteritis or enterocolitis. This may show colicky pain with vomiting, fever and collapse, but diarrhea is more frequent and the course will usually clear up the diagnosis. It must be borne in mind that this condition may give rise to peritonitis by extension through the lymphatics of the unbroken intestinal wall. In a case of peritonitis under the writer's care at the Cook County Hospital, a diagnosis of typhoid fever with perforation was made. Operation showed no atrium and, in the subsequent course, typhoid was excluded. It was a non-specific enteritis with secondary peritonitis.

Ruptured tubal pregnancy may give rise to difficulty, but the history, the physical signs on pelvic examination and the early developing acute anemia should clear up the diagnosis. Rupture of an abdominal aneurism, embolism of the superior mesenteric artery and thrombosis of the corresponding vein have led to doubt in a few cases. Each would show pain and an early collapse with abdominal tenderness, etc., and, in the last two, peritonitis may develop. A study of the cardio-vascular system should lead to the proper diagnosis. A serous diarrhea is a constant symp-

tom in thrombosis of the mesenteric vein. Hysterical peritonitis has been described, and Osler says it has "deceived the very elect." This would seem to be a case where to err on the safe side would be the part of wisdom, and these cases should be called peritonitis until proved something else.

Acute dilatation of the stomach, especially when postoperative, has led to confusion. Halstead[1] has recently reported such a case. The diagnosis would rest here on the stomach examination. Twisted pedicle of an ovarian cyst and torsion of an omental tumor have to be remembered as possible confusing conditions. Finally, it must not be forgotten that thoracic lesions may simulate peritonitis. Pneumonia, for example, may show pain in the abdomen and give rise to confusion. These conditions should, however, be cleared up by the thoracic examination.

Local peritonitis will, at times, be difficult of diagnosis. A subphrenic abscess, for example, may be confused with certain thoracic conditions as pleurisy or, more often, empyema and pyopneumothorax. The signs may be much the same, but the history of a thoracic condition on the one hand and of an abdominal condition, especially appendicitis, or ulcer of the stomach on the other, points the way to a proper solution. I have found that the preponderance of abdominal over thoracic involvement of especial value in this differentiation. An empyema may push the liver down, for example, but only when it is extensive and reaches high up, while a subphrenic process may extend upward, but only when its downward extension is marked. The findings on exploratory puncture will often be of value. Where pus is obtained by aspiration in one intercostal space and a serous fluid from the space next higher to it, subphrenic abscess must be thought of, though the same findings have been observed in pleurisy with localized empyema. Fürbringer noted that where the needle perforated the diaphragm it showed excursions more marked and in the opposite direction to those obtained from puncture of the pleural sac. Pfuhl has shown that the manometer may be of value in this differentiation. When the pus lies below the diaphragm the pressure is greater on inspiration, while the opposite holds in empyema. The *x*-ray may be of value in locating the position of the diaphragm. In this same condition, liver abscess may be thought of. Absence of jaundice and of tenderness of the liver would speak against abscess of that organ. It must be remembered that subphrenic abscess may follow liver abscess.

Local peritoneal inflammations in the pelvis will have to be differentiated from non-inflammatory lesions in that region, especially tumors. The history will be of value here, and a careful local examination will, as a rule, clear up the diagnosis. I have been astonished to find cases of pelvic inflammation with almost a total absence of pain and tenderness. This must be remembered if mistakes are to be avoided.

The diagnosis of the cause of peritonitis will often be easy, but may be of considerable difficulty. Where, for example, a diagnosis of appendicitis has been made and a sudden sharp pain in the right iliac fossa occurs followed by a subsidence of the local signs and the development of general

1. Halstead: Journal of Surgery, Gynecology and Obstetrics.

pain, tenderness and rigidity with signs of free fluid in the abdomen and where the general symptoms of collapse develop, the diagnosis of perforation with general peritonitis can be made with reasonable assurance. Where, again, a puerperal infection is followed by the symptoms of general peritonitis the diagnosis will be easy. In those cases, on the other hand, where the patient comes under observation with a well-developed general peritonitis, the diagnosis of the underlying cause may be of great difficulty. Here the history of attacks of biliary colic or of indefinite symptoms due perhaps to cholecystitis, cholangitis, etc., may justify a diagnosis of ruptured gall bladder. Or perhaps the history may bring out the occurrence of pains in the epigastrium which were dependent on the taking of food, or it may be hematemesis has occurred when a probable diagnosis of ulcer of the stomach can be made. Some cases will be obscure, however, till an operation has been performed and a few may remain obscure till cleared up by autopsy. I remember a case of cryptogenetic sepsis with peritonitis where the atrium remained a puzzle till the autopsy showed a pyonephrosis as the starting point. The urine examination should have cleared this case up, but by some mistake a negative result was recorded. In an obscure case of localized peritonitis in the lesser sac the examination of the stomach contents showed a carcinoma of the stomach to be present, and the autopsy showed an ulcerative cancer which had ruptured into the lesser sac. It has happened to a prominent surgeon that an exploratory incision in the lower abdomen had to be enlarged upward till the origin of the trouble was found in a perforated ulcer of the stomach.

Before leaving the consideration of non-specific peritonitis, a brief discussion of the diagnosis of typhoid perforation will be in order. The difficulties of this diagnosis are manifold. On the one hand, a case may show at autopsy marked extravasation of intestinal fluids where no sign of peritonitis was present during life, or, on the other hand, a well-developed sharp pain followed by tenderness, rigidity, etc., led to a diagnosis of perforation, but an operation disclosed only a few flakes of fibrin and no perforation. In a case recently under observation at Wesley Hospital, a sudden severe pain in the right side followed by vomiting led to a suspicion of perforation. The abdomen was distended markedly, the liver dulness decreased, but not absent. The pulse was increased materially. Leucocyte counts showed no increase, however, two counts a few hours apart being about 8,000 each. The patient was watched carefully, but he improved markedly after a few hours and recovered uneventfully. The diagnosis must remain in doubt. There was no hemorrhage—the bowels were moving freely. Perhaps the meteorism gave rise to increased peristalsis and caused the pain, or it is possible that here as above we had to do with a localized plastic peritonitis by extension. Pain usually accompanied by rigidity and tenderness was the most frequent symptom in seven cases of perforation occurring among 500 typhoid cases at the Cook County Hospital in 1902 which were studied by Dr. F. G. Harris, of Chicago, and myself. It was present in five cases, as was also vomiting. Obliteration of the liver dulness occurred in three cases, and free fluid in

the abdomen was noted in two cases. Hiccough was also noted twice. At one time much was looked for from the leucocyte count, but the findings are so inconstant as to be of no value. A positive and marked increase may, however, help. It must be borne in mind that general peritonitis in typhoid may be due to other causes than perforation. These are extension from the intestine without perforation, ruptured spleen and ruptured abscess of a mesenteric gland. For practical purposes these may be disregarded in diagnosis because they are far rarer than perforation. The question of the diagnosis of typhoid perforation may be summarized thus: The diagnosis must be made on the symptom-complex, since no one symptom is pathognomonic. Where a sudden sharp pain, usually in the right iliac region, is followed by vomiting, marked abdominal distention with loss of liver dulness, and where the pulse rate is increased and symptoms of collapse come on, peritonitis due to perforation is to be suspected. If in addition the leucocytes show a marked increase within a few hours the diagnosis may be even more positievly made. As a general rule for therapeutic purposes it is better to err on the side of the positive diagnosis than to fail to recognize the condition when present.

A word may be said here regarding the diagnosis of chronic forms of peritonitis. An exudative and an adhesive form have been described. The symptoms are, as a rule, not marked. There may be pain and tenderness and sometimes fever has been noted. The diagnosis is often made first at autopsy, particularly of the adhesive form. The differential diagnosis from tubercular and cancerous processes, on the one hand, and from cirrhosis of the liver and other forms of portal obstruction, on the other, is often impossible.

Of the specific forms of peritonitis the tuberculous variety is the most frequent. Anatomically we note a general miliary form with slight evidences of peritonitis—tuberculosis of the peritoneum—and a similar form with marked peritonitis—tuberculous peritonitis. Often the process is limited to a few miliary nodules about an intestinal ulcer, and between these extremes we have every variety. Frequently there are larger tubercles and enclosed pockets that are often filled with a purulent or caseous material while the omentum is usually thickened and contracted or rolled up as a hard mass crossing the abdomen about the level of the navel. At times the process gives rise only to adhesions between the intestines with no exudate. There are, as a rule, other tuberculous foci found at autopsy. These are especially tuberculous bronchial glands and tuberculosis of the other serous membranes.

The symptoms and physical signs of tuberculous peritonitis vary. Pain and tenderness are usually present. Vomiting occurs at times and diarrhea is very frequently found. It is often due to intestinal ulcers and then is severe and persistent. Meteorism varies with the type of the process. Signs of free fluid may be obtained, but, as a rule, the fluid is more or less circumscribed into pockets by adhesions. Tumor masses may be felt frequently. Most often it is the thickened omentum that is felt, but larger and smaller tumors may be felt anywhere in the abdomen. The signs of a chronic intestinal obstruction may be present. The general

symptoms vary. As a rule, there is at least a slight afternoon rise of temperature. The pulse rate is rapid, often out of proportion to the temperature. Ehrlich's diazo reaction is often present in the urine. There are often signs of tuberculosis elsewhere, particularly in the apices. Paracentesis abdominis may give valuable data. The fluid is at times hemorrhagic. The tubercle bacilli may be found, but oftener a positive result will only be obtained by inoculating guinea-pigs. Considerable interest has been aroused in the cytology of the fluid, and a lymphocytosis has been considered as of value in the diagnosis of tubercular peritonitis. Finally, tuberculin injections must be mentioned as valuable when sharply positive. I have, however, seen two cases where a chronic process became suddenly acute after the use of tuberculin. Both terminated fatally in the course of a few weeks.

The differential diagnosis has to consider certain groups of cases. In the group where a generalized exudate occurs other causes of ascites must be considered, especially cirrhosis of the liver. When the frequent association of these two diseases is remembered, the diagnosis, however, becomes easier or harder, as the case may be. In a case recently under observation at Wesley Hospital, tuberculous peritonitis was suspected in view of the presence of cirrhosis of the liver and lymphocytosis in the ascitic fluid. The abdomen, too, was not uniformly soft after tapping, but ill-defined areas of resistance were to be made out. The autopsy showed no tuberculous peritonitis, however. Those cases of ascites due to heart and kidney lesions will, as a rule, be ruled out easily. The cases of chronic non-specific exudative peritonitis mentioned above would best be diagnosed tuberculosis clinically. Often the pathologist has hard work proving that they are not.

In the group of cases where a local exudate gives rise to a tumor mass clinically, other tumors will have to be differentiated. Signs of tuberculosis elsewhere, history, age, etc., will help to make the case clear. It must be borne in mind that an actinomycosis of the intestine may be present. The peritoneum may be secondarily the seat of a carcinoma when the primary growth may be recognized in the stomach or other viscus.

In the group of cases where only adhesions are present with signs of chronic obstruction of the intestines, other causes of the latter, especially carcinoma, must be ruled out. Here, again, the history, age and other signs of tuberculosis elsewhere will be of value. The chronic non-specific adhesive peritonitis should be diagnosed tuberculosis clinically or a diagnosis withheld. The balance of probability is in favor of tuberculosis.

Peritonitis due to the pneumococcus of Fränkel-Weichselbaum has of late years attracted considerable attention. In a study of the autopsy findings in 171 cases of lobar pneumonia posted at the Cook County Hospital,[2] I found acute peritonitis noted in seven cases. In most of these there was only a slight plastic exudate present. The peritoneal involvement here is only a part of a general infection. In fact, two of the cases showed endocarditis also, and in one of the two pericarditis was present, too. Peritonitis due to the pneumococcus has been found in lesions

2. Kerr, Transactions Chicago Pathological Society, 1903, vol. v, p. 274.

of the gastrointestinal tract without pneumonia, and the organism must be considered a fairly constant inhabitant of the alimentary canal. It is known that it is usually to be found in the mouth. Ghon[3] considered the mouth to be the atrium in three of four cases of fibrinopurulent peritonitis following gastric lesions with perforation—ulcer in one case and carcinoma in two. The fourth case showed pneumonia due to the pneumococcus and preceded the peritoneal lesion clinically.

The importance of these pathologic data from the standpoint of differential diagnosis lies in the fact that abdominal pain in pneumonia is not always to be considered as a referred pain, but in some cases indicates actual peritoneal involvement. A few cases occur where the exudate is large enough to give rise to physical signs, and then only the history and course will reveal which is the primary process. In the group of cases following gastrointestinal lesions the bacterial diagnosis is only to be made by the examination of the exudate.

That the gonococcus gives rise to an adhesive peritonitis has long been known, and every one knows how frequently these adhesions occur. Of late years several cases of general exudative inflammation have been reported. One such I saw with Dr. A. Loewy, of Oak Park. Following a periurethral abscess of gonorrheal origin, the patient, a young man of 21 years, developed abdominal pain and tenderness. There were well-marked signs of free fluid in the abdomen. The intestines were tympanitic and moderate constipation was present. There was some fever. Subsequently all these physical signs disappeared and an uneventful but protracted recovery resulted. While a positive diagnosis is impossible here, yet the probability is that we had to do with gonorrheal peritonitis. It was at any rate a peritoneal inflammation by infection through the vascular system of a relatively benign type. A positive diagnosis in such a case can only be made by bacteriologic examination of the exudate. It is important to remember, however, the possibility that such processes may be of gonorrheal origin.

To recapitulate, we have considered the pathology of peritonitis, the symptoms and signs of acute general and local peritonitis and their differential diagnosis, the diagnosis of the cause of peritonitis, the diagnosis of typhoid perforation, and the pathology and diagnosis of the specific forms—tubercular, pneumococcic and gonorrheal peritonitis. The secret of success in making a diagnosis of peritonitis as of any other condition lies in the complete and comprehensive study of each case and in drawing logical deductions therefrom. A not inconsiderable group will be found where a clinical diagnosis without operation is impossible. The surgeon will settle some of these for us, but there will remain a few for the pathologist to clear up. The late Professor Fenger, than whom no more careful student and no surgeon of better pathologic training ever lived in the west, once operated on a case for perforation of an appendix where the autopsy disclosed general sepsis with peritonitis following vaccination. Such examples should prevent us from becoming too dogmatic in our diagnoses.

3. Ghon: Wiener Klinische Wochenschrift, 1904, vol. xvii, p. 267.

PATHOGENESIS, ETIOLOGY AND PATHOLOGY OF PERITONITIS.*

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The agreement between Dr. Tieken and myself is that I will discuss peritonitis, including the inflammatory accumulations of fluid, while he will discuss the non-inflammatory accumulations. As to the major portion of the etiology of peritonitis, there is but little room for discussion. The following points will be stated without argument or explanation.

The exciting etiologic factors in peritonitis are:

1. Any of the pus-producing bacteria habitually resident in the gastrointestinal canal or capable of securing easy entrance thereto.

2. Any of the facultative parasites, such as colon bacillus, which are habitually or temporarily resident in the gastrointestinal or genitourinary tracts.

3. Any of the parasites capable of exciting inflammatory reactions, non-suppurative in character, e. g., tubercle bacillus.

ROUTE OF INFECTION.

Two are of consequence. In order of importance, they are: 1, The genital tract in the female; 2, the gastrointestinal tract; 3, the urinary tract; 4, adjacent cavities, e. g., pleura; 5, adjacent tissues, e. g., injured fascia, perinephritic abscess, caries of the bone; 6, blood stream infection.

Peritonitis is usually an infection by the lymph route. It is seldom a blood stream infection. The reason for this I am at a loss to understand. For example: of the pleurisies, with relatively acellular effusion, 56 per cent. are tubercular. Most of these are without concomitant lung lesion. In the last 1,000 autopsies made at the Cook County Hospital, there were small pleural and subpleural healed tubercles in 283. Tubercular peritonitis, in the first place, is quite rare. In this series of autopsies already referred to, there were 33 cases, 3.3 per cent., of miliary tuberculosis with involvement of the peritoneum, of which 16, or nearly one-half, showed tubercular ulcers. The last case of miliary tuberculosis, posted by me two weeks ago to-day, showed miliary tubercles in the lungs, liver, pericardium, spleen, kidney and peritoneum, accompanied by scores of tubercular retroperitoneal glands, the largest of which were 5 cm. in diameter. In this case, there was no break in the mucous membrane of the gastrointestinal tract or the adjacent tracts. The possibility of infection of the adjacent lymph ways by tubercle bacillus through an intact mucous membrane is generally accepted at the present time. Cummings, in a study of 3,405 autopsies at the University of Pennsylvania, found infection of the peritoneum in 92, or 2.7 per cent. or 11 per cent. of all cases of tuberculosis. Borshke, in 4,250 autopsies, found it in 226, 5 per cent.

* Read before the Section on Medicine, Illinois State Medical Society, at Springfield, May 15-17, 1906.

We hear so much of the genito-urinary route of infection of the peritoneum in females that I have been surprised to find the relative infrequency of tubercular peritonitis in the female. In the estimate of the importance of different routes of infection of peritonitis, the genital route in the female was placed first. This was in consequence of the fact that 70 per cent. of all peritonitis is suppurative in character and due to one of the habitual pus organisms or a bacterium, such as coli, capable of becoming a pus producer. According to Nothnagel, 24.8 per cent. of all peritonitis is tubercular. Cummings' figure is 25 per cent. Cook County Hospital autopsies do not give bacteriologic data. About 5 per cent. of all cases of peritonitis is due to subinfection, continuous leakage of mild bacteria, eventuating in serositis, porcelain peritonitis, etc. This leaves 5 per cent. for all other forms. In this 70 per cent. group (the pus infections), the female genital tract ranks higher than the gastro-intestinal tract or any other.

In the 25 per cent. group (the tubercular infections), we are confronted with the following figures: Osler says that of tubercular peritonites 70 per cent. are in males. Vierodt says 80 per cent.; Cummings, 65 per cent.; Sick, 80 per cent. If we were to assume that the female was equally liable to contract tubercular peritonitis through her gastrointestinal tract and through other routes, we would be justified in contending that her genital apparatus serves to protect her in some measure against this infection. It would not, then, seem to be a matter of enormous moment whether the tubercular salpingitis, which we find in these cases, precedes or follows the peritoneal infection.

In a series of experiments, we found that when virulent tubercle bacilli were injected into the peritoneal cavities of monkeys the major infection was found in the dependent portions. In female monkeys, walking upright, it was found in the pelvic peritoneum and especially around the Fallopian tubes.

In the entire series of 1,000 autopsies at Cook County Hospital, tuberculosis was present in 481, or 48.1 per cent. Of these, 163 had ulcerative consumption and, of these, 76 had tubercular intestinal ulcers. Of the 283 cases in which the tubercular nodules were recorded as healed, there were 12 cases with tubercular intestinal ulcers. When these are added to the 16 cases of ulcer associated with miliary tuberculosis, we have 104 cases of tubercular ulcer of the intestines. These figures demonstrate a tolerance of the peritoneum for tubercle bacilli, a tolerance which the surgeon had previously discovered. It is with him an axiom that some cases of peritoneal tuberculosis with ulcer recover after exploratory section, whilst most cases recover in those cases, where there is no ulcer.

Five per cent. of peritonitis is of the serositis type. That is, the subinfections with intestinal bacteria of a very mild grade of virulence. As these cases are not recognized clinically, unless they are widespread, I think that the percentage is very much higher. In fact, it has been my experience that every adult coming to autopsy shows some evidence

of bacterial leakage in his abdomen. In a study of the last 3,550 autopsies at Cook County Hospital, periappendiceal adhesions were found in 486, or 13.6 per cent. Comparatively few of these adhesions were due to suppuration infection. Most of them were mild progressive productive inflammations of the pericecal and periappendiceal tissues due to bacterial leakage. It has been my experience that there are other regions of the abdomen in which these adhesions are still more frequent. Where the splenic flexure crosses the psoas muscle, such adhesions are almost habitually encountered. Adami and his school were right in recognizing the etiologic relations of subinfection. In the main, Byron Robinson was right in ascribing etiologic importance of the physical factors contributed by the contraction of the psoas muscle. Let us keep clearly in mind that this periappendicitis is not and has not been suppurative appendicitis, and the finding of such adhesion bands does not mean that there has been previous attacks of suppurative appendicitis. However, such adhesions are determining factors in suppurative appendicitis. The importance of appendicitis comes not from the infection, but from the difficulty of drainage of this narrow tube, anatomically so arranged that, when it swells, it must swell inwardly. Anything which compromises the physics of this organ is of major importance, because the physics of the appendix is paramount. Such adhesions may determine suppurative appendicitis. The physical factors I have considered in a paper on stricture subsequent to intestinal anastomosis, read before the American Medical Association. The points there made were that strictures developed in the intestine only at those points where the tube was bound, so as to prevent longitudinal movement; that even annular ulcers, in which there was a good deal of scar tissue from infection, did not develop strictures, unless the ulcer was situated at some fixed point, such as the rectum, the fixed points in the colon, etc. The scar connective tissue fibers, running always in the direction of pull, would not be found circularly arranged where there was longitudinal or vermicular movement.

The localized growth of subendothelial adhesions, due to subinfection, is seldom of consequence, except around the narrow tubes, as the appendix, the Fallopian tubes, the gall tract and possibly the pancreatic ducts. In consequence, localized serositis is overlooked in autopsy reports, except in these localities. That it is present in most adults has been my autopsy experience. When it becomes more general, spreads to the convexity of the liver and spleen, the parietal peritoneum, the adjacent serous cavities, we have to deal with polyserositis, a definite clinical entity, but one that is usually overlooked clinically. The lesions of these infections are usually described as occurring in the upper abdominal segment, thus agreeing with MacCallam's opinion that the route of travel of substances in the peritoneum is toward the central tendon of the diaphragm. The polyserositis lesions are most frequently found on the anterior surface of the retroperitoneal space on the convexity of the liver and spleen and on the under surface of the diaphragm. But few are noted anteriorly or inferiorly in the abdomen. Why these subinfections travel up and tubercular infections settle down into the pelvis is diffi-

cult to understand. It must be because the very mild bacteria are allowed to flow along in the physiologic way, whilst barriers are placed for those which are more virulent. Yates has shown that any pus-infected area of the peritoneum, unless lytic bodies are very active, is made extraperitoneal in about six hours, whereas relative encapsulation is immediate. I can only suggest that in subinfection the bacteria (e. g., colon) are allowed to flow along the physiologic route. The milder forms of pus cocci and the non-virulent forms of tubercle bacilli are encapsulated and made extraperitoneal, relatively so in a few minutes, absolutely so in a few days, with much individual variance. The more virulent forms of bacterial infections, especially suppurative bacteria, are lytic to any encapsulative material and, therefore, freely spread along the physiologic absorption routes.

DISCUSSION ON THE SYMPOSIUM ON SEROUS INFLAMMATIONS OF THE THORAX.

Dr. E. F. Ingals, of Chicago:—Mr. Chairman: Dr. Taylor spoke of several punctures being made and the failure to find pus. I do not think that there was any particular harm in making these repeated punctures, and I thoroughly agree with him. It occurred to me, however, that in the majority of the cases that require several punctures it is not empyema but a pulmonary abscess that we have to deal with, and there is no objection to hunting for the abscess.

He said that when the empyema is discovered there should be immediate drainage. I do not wish to take exception to that statement, though I do not exactly agree on the time of the withdrawal of all the pus. I think it is much better to drain promptly, but not completely. We should withdraw as much of the pus as we can without causing uncomfortable symptoms, but when the patient begins to cough or has a feeling of weight and pressure on the chest which is possibly attended with a good deal of pain, we should cease aspirating at once. I do not favor opening the chest radically at this time in most cases, but I would repeat the aspiration again and again every four or five days until the chest had been entirely emptied before making the larger opening for free drainage. I think we get better results in this way in the majority of cases as the lung dilates more rapidly.

One of the essayists in speaking of the physical signs of pleurisy, spoke of vocal fremitus. I want to emphasize the value of this sign in distinguishing between fluid and a consolidated lung. Many of the points that he brought out are often not even thought of by the physician, yet they are very important; but this particular sign should be emphasized for the benefit of the younger men of the profession.

I was greatly pleased with the last speaker's paper, because it seemed to me to show clearly what the consensus of opinion would be among competent observers after long experience. This address was not a compilation, but the record of personal experience. I think most of us who have practiced for some years have had the same experience. I am heartily in accord with what was said about the various pastes. I do not order them, but if people want to use them I do not object. The application of heat, by means of the hot water bag, was not mentioned. The Leiter coil may be used with hot water as well as cold. Neither was the oiled silk jacket mentioned. I speak of these because one of the other speakers objected to poultices, claiming that they are in the way of examining the chest. The oiled silk jacket should be made in two pieces, pinned together at the sides. A layer of cheesecloth should come next to the skin, then oiled silk, then two inches of cotton, and then some more cheesecloth. This will hold the heat as well as a poultice. It does not require frequent renewal. It is easily lifted from the

chest, and the heat can be increased if necessary by laying a hot water bottle against the chest. In the thirty-five years that I have practiced medicine I have never seen any good come from the use of tincture of iodin applied to the chest at the seat of pleurisy. In the treatment of serous exudates which have lasted a considerable length of time, the question arises as to when we should aspirate. I have always instructed students that the serous exudates, excepting where there is unusual pressure, should not be withdrawn for ten or fourteen days, because the serum is there for the purpose of keeping the two inflamed surfaces apart. When the exudate remains longer than two or three weeks without diminishing decidedly, I am accustomed to direct that it should be withdrawn. My experience has not been the same as the last speaker's with reference to pleurisies that may have been of tubercular origin. I have not seen ill effects from the removal of fluid after it had been present for three weeks. The statement has been made that aspiration causes tuberculosis in the lung to increase. Possibly this is true in rare cases, but I am confident that the number of cases that would be greatly benefited by the removal of the fluid is very much larger than the number that would be injured. As to the prevalence of tuberculosis as a cause of pleurisy, I think that the belief that nearly all cases of pleurisy are tubercular is wrong. If there is tuberculosis in the lung then we may take it for granted that a concurrent pleurisy is tubercular, but if not, then it is necessary to demonstrate the presence of tubercle bacilli in the fluid either by the microscope or by inoculating guinea-pigs before we are justified in attributing the pleurisy to tuberculosis. Dr. Bowditch traced the history of 250 of his father's cases that were aspirated for pleurisy, and he found that within fifteen years 50 per cent. of these patients had pulmonary tuberculosis. Inasmuch as nearly every one has pulmonary tuberculosis at some time (97 per cent. of all adults, according to Wageli's figures), it would not be strange that 50 per cent. of those patients should have it within fifteen years, even if tuberculosis had been the cause of the pleurisy in only a very few. I think it is an error to assume that pulmonary tuberculosis is a large factor in the etiology of pleurisy.

Dr. Arthur R. Elliott, of Chicago:—I have but a single comment to make, and that is with regard to Dr. Davis' statement relative to the treatment of pulmonary effusion. He advocates waiting until two weeks have elapsed, or until evidence is at hand of the persistence of the effusion. He rather discouraged the resort to paracentesis in pleural effusion of subacute tubercular origin. Some years ago Dr. Austin Flint, Jr., advocated very strongly the early withdrawal of pleural effusions in all cases in which the effusion was sufficient to cause displacing of the heart, with much increase of the intra-pulmonary tension, and with resulting increased labor to the right heart. He reported a series of cases, which were certainly very strong proof of the efficiency of this practice. Since then I have made this my guide, and I perform early paracentesis, repeating it in all cases in which the effusion is considerable, and, especially, when the heart is displaced, repeating it as often as is necessary to keep the pleural cavity free from accumulations of fluid. I insist on this particularly when there is any evidence of cardiac trouble or nephritis, where pleurisies are apt to occur. I have come to dread the presence of a pleural effusion, in cases where there is a bad heart. I draw the fluid off early under the strictest aseptic precautions, because it limits the formation of adhesions and takes the strain off the heart.

Dr. Arthur Dean Bevan, of Chicago:—I have been asked to discuss the surgical treatment of peritonitis. In the discussion of this question we must discuss three methods of treatment: The saline treatment, which has been advocated especially by gynecologists; the rest and opium treatment, which has been advocated especially by the internists, and some surgeons; and the operative treatment, which is that of the surgeon, as a rule.

The saline treatment of peritonitis is really an impossible one from some standpoints. There can be no doubt, as shown by Day, that in some cases where

the symptoms would lead one to believe that we have to deal with a peritonitis, and where it really may have existed, the employment of salines to the extent of eventually producing catharsis has been abandoned. However, one must analyze very carefully the conditions which are present in these cases. The cases where the saline method of treatment of peritonitis is of value are cases in which we have to do with a partial paralytic ileus, and where the danger to the patient is not from absorption to the peritoneum, but from absorption to the mucosa. In other words, where this method is of value, is only in freeing the alimentary canal of toxins which would be absorbed by the mucosa and produce very serious and some times fatal results, as are produced, for instance, in cases of ileus. Patients frequently die from ileus without any peritonitis as the result of an intestinal toxemia. So that in many cases where there is this partial ileus the so-called saline treatment of peritonitis is of distinct value. Of course, it has been shown by autopsies *in vivo* that in a general way the saline treatment of peritonitis is of no value whatever.

In the same way one can speak of the rest and opium treatment of peritonitis. It is of no value in cases of general suppurative peritonitis, but is of distinct value in cases of circumscribed peritonitis. Without doubt, many cases of peritonitis, which are limited and circumscribed, are prevented from becoming general by the opium and rest treatment, and, as a general proposition, in the cases of appendicitis, in which the conditions surrounding the patient are such that he can not be given the benefit of a better treatment, i. e., the removal of the focus of the disease, the opium and rest treatment is advisable. Frequently, some organic lesion or the inability to give the patient an anesthetic, so that operative intervention is out of the question, makes it better to handle these cases by this method than by the operative treatment.

When we come to cases of general suppurative peritonitis, there is this to be said, that the operative treatment is largely prophylactic. The case that is saved is the case that is operated on early, the damage done to the alimentary canal repaired so early, the noxious fluids which are in the peritoneal cavity removed so early, the peritonitis never results. The cases which appear in the literature of gastric perforation that are operated on in a few hours, with the perforation controlled and the leakage prevented by a few pursestring sutures, those are the cases where peritonitis is actually prevented from developing, as a rule.

Now, we go a step farther, and we have the cases of general suppurative peritonitis complicating gastric ulcer or appendicitis, where the noxious material has been in contact with the peritoneum for hours. Here the operative treatment of the modern surgeon shows wonderfully better results over the operative treatment of five years ago. What was it five years ago, when McCosh, Senn, and others, discussed this treatment before the American Surgical Association in Washington? It was evisceration of the patient. The intestines were exposed, turned out on a hot towel, and the pus was wiped off from the surface of the peritoneum, with the result that the majority of these patients died. In these cases, to-day, without a bit of exposure of the intestines, with the finding of the focus which is responsible for the peritonitis, and with the prevention of further leakage by proper suture, we wash out all the noxious material from the peritoneal cavity, instituting drainage or not, as may be required. The great majority of these patients recover. I can say that from my own personal experience. All the cases of general peritonitis which we have had in the last three years have been handled successfully. I mean typhoid perforations, gastric and duodenal ulcer, and appendiceal perforations. We look for the focus, control the point of leakage, wash out the noxious material, and then suture. Take, for instance, a duodenal ulcer with perforation. You find on opening the abdomen an ulcer high up and a limited amount of material about it. How do you know whether the entire peritoneal cavity is full or not? Or take a perforated appendix. You expose the appendix and find a little fluid about it. It is a question whether there is a gross

involvement. The method of exploring the entire abdomen with a female glass catheter, introduced through the wound into the cul-de-sac, placing the thumb over the catheter, and then withdrawing the latter, will surprise you in the manner that this glass catheter will test every part of the abdominal cavity. If there is a dram of pus in the cul-de-sac, the catheter introduced into it will draw out a large part of the pus. In the same manner you can test the region of the liver. If there is a fluid, wash it out, and then put in a small cigarette drain through a counter opening above the pubes down to the appendix.

For three years Blake has been doing away entirely with these counter drains, making immediate closure. He finds that his results have been much better than they were in cases where he had established drainage. I have not yet been willing to dispense entirely with the drainage, but certainly the success we have had in the last five years warrants us in saying that the great majority of cases of general peritonitis, the result of perforation, if operated on in the first twelve hours, can be saved, but they can not be saved in any other way than by surgical intervention.

Dr. A. E. Halstead, of Chicago:—It is impossible to discuss the surgical treatment of peritonitis without taking into consideration the bacteriologic cause. There are certain types of general peritonitis that do not belong to the surgeon. In this class surgical treatment is not indicated, because the patients die whether operated on or not. I refer to the type of diffuse septic peritonitis, the result of a streptococcic infection, where there is little or no exudate, but the entire peritoneal surface is inflamed. No treatment is of any avail. My results in general suppurative peritonitis have not been as good as those of some other Chicago surgeons. I can not cure 75 per cent. of the cases I see. I have come to a conclusion, that, in cases that do get well, the drainage has been of little practical value.

I want to emphasize the great necessity of removing the cause. A few years ago we were taught that all that was necessary, in general peritonitis, was to open up the abdomen and drain, without looking for the cause. Now, we invariably search for the cause of the peritonitis and remove it if possible. It is always removable if due to a perforation of the appendix or an ulcer.

As to the value of drainage, we must realize that within a few hours after we place the drain in the abdominal cavity, the drainage channel is shut off from the peritoneal cavity and the only space you are draining is the part occupied by the drain, and that the drainage of this small cavity can in no way affect the inflammation that is present in other parts of the abdominal cavity. It is difficult to see how any treatment that we can institute, other than the mechanical removal of the pus and fluid, can be at all effective. I believe that the patients that recover from general peritonitis are those who have a very mild form of infection of some kind. I do not consider that any intense infection, if distributed over the whole peritoneal cavity, is susceptible to any curative treatment, except in a few cases where the resistance of the patient is great or the infection is of a mild type. I have had a considerable experience in the treatment of general peritonitis, and I consider it more good luck than good surgery when my patients recover. I am not speaking of cases of local peritonitis where only a small portion of the peritoneum is involved. In those cases, if you remove the cause, sponging out the part involved, the patients will get well. But in the cases where there is a general infection of the whole peritoneum, the prognosis, from my point of view, is extremely unfavorable.

Dr. W. A. Evans, of Chicago:—The first essential of the medical treatment of peritonitis is to find the route in which the infection has occurred, and to remove that source of infection when this is possible. I believe that that as a factor in the treatment of peritonitis should never be lost sight of; and so far as this consideration carries we should never let the issue be doubtful by contemplation whether this is a medical or a surgical case. I tried to make plain in my

paper that peritonitis was usually an infective disease, in which the route of infection was some one of the adjacent viscera, and it is not infrequently due to primary infection, in some adjacent viscus, which can be located and which can be operated on. Therefore, whether we call it the medical or the surgical treatment of peritonitis the paramount duty of the diagnostician is to locate the atrium of infection and to determine as best he can what is possible in the way of eliminating this atrium.

The next question that must be answered is, what is the proper medical treatment of those cases in which, for one reason or another, the case must be treated wholly or in part as an internal disease. I believe that there are certain cases of peritonitis in which a certain amount of exhibition of opium is desirable, just as there are certain cases of cough, of vomiting, of constitutional perturbation of one sort or another in which constitutional coma, for a few hours at least, is desirable, but I believe that so far as the regular routine treatment of a single case or of a considerable number of cases is concerned, that the use of morphin in the treatment of peritonitis is of limited value, and usually does harm.

I believe that there is no specific treatment for peritonitis at the present time. For that reason there is necessity for more of medical treatment than in those diseases where there is possible a specific treatment. You can all remember the time when you were called in on a case of diphtheria, and you had to carefully nurse and treat the patient through two or three weeks. Then there came a time when there was found a specific, and all that was necessary for the physician to do was to go and see the case, make his diagnosis, and inject antitoxin. A number of years ago Dr. Bevan was quoted as saying that there was no treatment for pneumonia. The conclusion was drawn at once that there was no place for the doctor in the handling of a case of pneumonia. The point he wished to make is that in these diseases where there is no specific medication, there is increased demand for medical supervision and treatment, the treating of symptoms and conditions as they arise.

I wish to advocate in these cases the saline treatment and to emphatically disagree with most that Dr. Bevan said on this proposition. The saline treatment I now refer to is the continuous injection into the rectum of saline solution, with an apparatus so devised that there is a continuous slow flow of the solution into the bowel, a flow that is just as rapid as absorption is possible, so that there is no escape of the fluid from the bowel. It is poured into the general circulation and is eliminated in part by the skin and in greatest part by the kidneys. These people die from toxemia, because their nervous system is affected. It is not possible to wash out the peritoneal cavity by any method of drainage devised, and any surgical procedures directed along this line have been abandoned. But it is possible to wash out the general blood supply with salt used in the manner indicated, and I have seen cases of sepsis, puerperal sepsis, and other forms of peritoneal infection, in which there have been marvelous results obtained by this continuous rectal injection of salt solution.

Dr. Channing W. Barrett, of Chicago:—The division of the treatment of peritonitis into that of surgeon, internist, and pathologist is most unique. Had the previous speakers divided peritonitis into pelvic peritonitis and perforative peritonitis, the former falling into the hands of the gynecologist and the latter into the hands of the surgeon, the treatment of the one having no great tendency to becoming extensive, being slow in onset and usually limited to the tubes, having no great tendency to immediately kill the patient, but to destroy the pelvic organs, then I might have spoken of the treatment by the gynecologist. The gynecologist can be something of an abdominal surgeon, and has acquitted himself fairly well as such. Therefore, instead of dividing these cases into cases to be treated by the general surgeon and cases to be treated by the gynecologist, they should be divided according to the nature of the case. If the surgeon gets hold of the pelvic case, we hope he will treat it well. We drain pelvic peritonitis much quicker than we did formerly. We open the posterior cul-de-sac early and drain. Thereby we get a better condition of the tubes afterward.

DISEASE IN THE AGED.*

W. H. Curtis, M. D.
WILMINGTON.

It was with a becoming degree of hesitancy that I acceded to our chairman's request for a paper upon disease in the aged, because profoundly conscious of my own inadequacy to properly perform the task. However, my connection with one of the state institutions, maintained for the care of this class of patients, having afforded me, perhaps, a better opportunity for observing the processes of disease in the later decades of life than is given to most of you, as it certainly has afforded me abundant opportunity for recognizing the urgent need of a more thorough and exhaustive study of these processes, must serve as my excuse for this paper, which I trust may, at least, help to excite your interest in its subject, even if it fails to instruct.

The senile individual would, *a priori*, appear to offer peculiar attractions for the scientist and therapeutist, for, according to modern ideas, senility is but the incarnation of disease itself; in other words, the weight of evidence seems to establish the fact that old age is never physiological but always pathological—at least its visible and appreciable evidences are pathological ones. But what are the facts? If one turns to his library for some authoritive work upon the diseases of this period of life, unless peculiarly fortunate, he is doomed to disappointment. Inquiries made of our medical publishers for an up-to-date work upon this subject have, with me, proved unfruitful, but their replies have invariably been accompanied by announcements of innumerable recent works upon the diseases of infancy, adolescence, and adult life.

It is true there exists an extensive literature upon the subject, but it is so widely diffused as to make it practically unavailable. In this day of many specialists it may well be asked why this should be so. Is it because we look upon the aged as we do upon an outworn garment, not worth the mending? This view hardly appears creditable to our philanthropy, even were it possessed of economic value, which it is not; for many a mentality of eighty is of infinitely greater value to the state than is the mentality of twenty. What might we not have hoped for from the genius of a Humboldt, a Faraday, a Pasteur, could our art but have prolonged the span of their usefulness but a few more fleeting years? And the pity of it is we know this very desirable result might well have been accomplished.

This may sound like mere unwarranted assertion, but I think you will agree with me that it would not prove an arduous task to formulate a plan of life, by the observation of which a race of individuals might be reared, whose span of life might confidently be expected to reach the norm of five score years; and it is encouraging to know our theories upon

* Read before the Section on Medicine, Illinois State Medical Society, at Springfield, May 15-17, 1906.

these subjects are slowly materializing, the average age of the individual is gradually growing longer. But it must be observed our most notable results in this field have been obtained in the periods of infancy and adolescence, and should not be taken as applying to all periods of life. I have not reliable figures at hand pertaining to our own country, if such exist, but in Great Britain, since 1859, the death rate in all ages under 55 has declined 17.5 per cent, only 2.7 per cent, however, in ages above 55, while, between 65 and 75, there has been an actual increase.

Investigations have shown that only some nine hundred in the million die a natural, physiological death, fall as does the fully ripened leaf; all others come to their graves by processes strictly pathological and unnatural. These few figures would seem to indicate that we are not doing our full duty by "the old folks."

The pathological processes resulting in senility are now quite well understood, as are the methods for their prevention; but, as I have already intimated, this knowledge is so widely diffused throughout medical literature that its usefulness to the general practitioner has been greatly restricted. As the etiology of disease in the aged frequently antedates the advent of years, it is manifestly impracticable to fix upon any hard and fast rule by which to determine whether any particular number of individuals are old or young; this can only be determined by their individual pathology, an accurate estimation of which will frequently disclose the fact that one individual is old at twenty, while another at eighty is still young. Probably as good a working rule as any is the well known axiom "a man is as old as his arteries," and this thought was apparently recognized by the poet long before its adoption by the profession in its axiomatic form, when he said: "We measure time by heart throbs, not by figures on a dial." It has of course long been recognized that certain changes in the circulatory system were the almost constant accompaniment of diseased processes in the aged, and sometimes, too, it has been recognized that these changes were apparently the direct and immediate cause of the disease. But unfortunately, the idea has too long been entertained that this hyperplasia of the arterial coats was the inevitable consequence of advancing years, in other words, a normal, physiological process. When in time we learned our error, search was instituted for the cause of these pathological changes. Reasoning from analogy and clinical experience, it was soon proclaimed that syphilis was the etiological factor in fifty to seventy-five per cent of all cases of arteriosclerosis. Then alcohol had its day in court, and was thought to even outrank syphilis as a causative factor. Lead, mercury, and other mineral poisons have all had their champions, but, as might have been foreseen, none of these were found applicable in all cases.

As our pathological knowledge increased, our etiological boundaries widened, and while it appears to be firmly established that this condition is always the result of poisoning, either introduced from without or generated within, I think it is now generally recognized that not one but

many poisons, be they mineral, vegetable, leucomain, ptomain, or what not, may be and are the cause of this condition. The profession is to be congratulated if this knowledge shall contribute to help us to forever renounce our worship of the fetich of disease, *per se*, and bring us back again to the fold of our fathers, who taught us the concentrated wisdom of medicine in the axiom "generalize your disease and individualize your patient."

If, then, arteriosclerosis is to be accepted as the primal factor in the diseases incident to old age (and without doubt it may be so accepted), it naturally follows that it is eminently essential that a most thorough and exhaustive investigation of the evidences of this condition be instituted, in those cases where it may be suspected to exist. While exceptional practitioners do employ modern methods for the determination of this question, I am persuaded that the great majority do not. As the distinctive symptom of this condition is admitted to be the pulse of high tension, it is permissible to enquire how this high tension is determined to be present or absent. There may be a few skilled diagnosticians, whose tactus eruditus is so abnormally developed that they can detect the very onset of this condition, or, at least, early enough to render their therapeutics of avail. But this almost superhuman skill is not vouchsafed to most of us. At that late stage of the pathological process when the radial artery rolls like a whip-cord beneath the finger, when the left ventricle has become hypertrophied to twice its normal thickness, and when one can observe the throbbing of the carotid across the room, then even the tyro can make the diagnosis. But when this stage is reached, the possible results of effective therapeusis are enormously diminished.

We have, however, an instrument of precision, not so generally employed as I think it deserves to be, which will usually enable us to determine the advent of this condition in ample time to render effective therapeutic aid. I allude to the sphygmomanometer, very many observations with which lead me to regard it as fairly accurate as between individuals, and quite reliable as to different readings in the same individual. I am convinced that the more general employment of this very useful diagnostic aid will not only prove of great benefit to a rather large class of patients, but that it is also liable to contribute something in the nature of a surprise to many a practitioner, when he thus, for the first time, discovers the unmistakable evidences of senility in those still young in years.

My observations have led me to conclude that there is a somewhat numerous class of persons in the middle decades of life who exhibit such symptoms as languor, lassitude, defective memory, perhaps headache and irregular breathing, with possibly vertigo, etc. In short, they present the symptom complex of neurasthenia, and as such they are, I think, usually diagnosed. When subjected to the proper test they will be found to have a pulse of high and increasing tension, and are, therefore, really cases of incipient arteriosclerosis. The correct diagnosis of these and similar cases opens up a broad field for effective therapeutic effort, in-

cluding the prevention of many secondary affections, such as heart and kidney diseases, neuroses, etc.

But it must not be too hastily concluded that even in advanced cases of arteriosclerosis, such as are so frequently seen in those advanced in years and evidenced by the rigid artery, hypertrophied heart, defective elimination, etc., that nothing can be done for their relief. Given a patient old in years and old in his arteries, with an hypertrophied heart that staggers under its weary load, with veins surcharged to bursting, bodily functions in practical abeyance, respirations hurried and irregular, mentality a comparative blank, their rising up a sorrow and their lying down a torture hardly to be borne—what of these?

That the picture is not overdrawn you know, for you have seen it frequently; that it is one before which we need not despair I think I know, for, during the past ten years, I have had a hundred such pictures under daily observation.

I do not pretend to be a worker of miracles, medical or otherwise, but I do wish to say that suitable environment, appropriate attention to diet, functions, clothing, exercise, etc., in short, the observation of the whole train of hygienic virtues, and, above all, the lifting of the load from the overweighted spring will surely result in the restoration of at least a portion of its lost elasticity. The attainment of desired results in senile disease is attended by difficulties not met with in those in whom metabolic changes are, for physiological reasons, more quickly and easily accomplished; but those cases are rare in which Nature is not willing to meet the physician at least half way. Indeed, the possibilities for partial if not complete restoration in these outworn machines frequently appears to border upon the marvelous.

In this connection I desire to say that I think our text-book dictums concerning these matters need revision. Old age and acute disease do not invariably spell death, as we are so frequently taught to believe.

As an illustration of this I would call your attention to the almost axiomatic teaching that pneumonia in those over 60 justifies a fatal prognosis. No one man's experience can determine a question like this, but my personal observation, given for what it may be worth, is that pneumonia, *per se*, is much less fatal in the old than it is in the young and middle aged. In the institution with which I am connected, with its one hundred inmates of an average age of 84 years, we have had within the past ten years seventy odd cases of pneumonia in its varying types, with no mortalities. I do not mention this at all boastfully, for I realize that it may be explained by the law of coincidence, but as this experience corresponds in the main with that of some 3,500 cases seen in private practice I may be excused for expressing the belief that pneumonia is not always nor necessarily a fatal disease in the aged. That it may be made so, by inappropriate or excessive treatment, is, I think, indisputable.

I spoke a moment ago of the difficulties attendant upon the treatment of the senile patient; these are many, and are not all to be accounted for by gross pathological changes. One of the difficulties with which I have had to contend has been the pernicious habits and modes of thought that

are the outgrowth of and have become fixed by time, in other words, the personal equation. This difficulty is not limited to any age, but it seems to attain its maximum in the aged. Every "old granny" has her infallible remedy for every ill, and many a brilliant medical reputation has come to wreck upon this uncharted rock. Fetich worship is still extant, and though it take the form of goose-oil, skunk-oil, or catnip tea, woe betide the young graduate who lays ruthless hand upon their altars. Probably the greatest single evil in this particular with which we have to contend is the widespread and altogether damnable habit of nostrum taking; to which pernicious habit more deaths among the aged are due than to any other three causes. These observations are not novel, and apply well to all periods of life, but seem peculiarly applicable to the period of old age, when we are called upon to deal, not only with the degeneracy of tissue, disease itself, but with the ultimate results of long years of wrong living; fixed habits of thought; prejudices, wrong perhaps in their inception and very essence, but almost or quite ineradicable in their dominant growth; perversions of function and of volition; enfeebled mentality, with all its train of attendant evils; together with scores of other evils and obstacles not met with in earlier life, all dominated, perhaps, by a strong and abiding faith, amounting at times to a real obsession, in the efficacy of the medicine bottle to again restore to them their lost youth. For these and many other cogent reasons, it is evident the diseases of no period of life present so difficult a problem for solution as do the diseases of old age; but as a complement thereto none appear to present a more interesting and fruitful field for study. As the difficulties attendant upon the ideal treatment of disease in the aged are so numerous, it naturally follows that the practitioner is called upon for the exercise of infinite tact and patience. Preconceived theories, even well grounded facts have frequently to be modified, even subordinated to individual idiosyncrasies. No two individuals travel through life by the same route, and the needs and requirements of each individual traveler are as variable as the vicissitudes of the weather, as changeable as the landscape upon the way; the "guide, philosopher and friend" of these old travelers must, therefore, oftentimes be content to travel their route, not his own.

I have had very little to say concerning the use of drugs in the diseases of the aged, not because I deem the subject of minor importance, but because I esteem it of such vast importance that at least a volume would be required to properly dispose of it.

However, I can not deny myself just a word or two in this connection. Our text-books are not very explicit in their expositions of the drug treatment of senile disease; the most significant general rule laid down for our guidance being, perhaps, that, in this extreme of life as in the other, our dosage should be greatly diminished. While perhaps correct, in the main it will be found wrong in practice in many instances, especially if the drug be administered per orem. Indeed, many conditions appear to demand enormously increased dosage to secure desired effects. I pre-

sume this to be accounted for by diminished power of absorption, lessened cell activity, etc. Libraries have been written upon the subject of the senile heart, the heart with an hypertrophied left ventricle and an accentuated second sound; its weaknesses, its shortcomings, its fragility has been pictured to us in language most vivid and convincing. Notwithstanding all this, I wish to say that a somewhat intimate acquaintance with this much abused but very tolerant organ, extending now over a period of nearly forty years, has been productive only of sentiments of profound admiration and respect. Properly conserved, I regard the senile heart as the most wonderful illustration of endurance the world can afford. Whipped and spurred to herculean effort for untold years of ceaseless labor by the steadily narrowing lumen of the peripheral vessels, it has thrived under punishment, grown literally great under conditions the most adverse, and now, in its old age, with its audible and piteous plea for rest,—what is our reply? It has been estimated that nine-tenths of the profession habitually employ digitalis in all conditions of so-called heart-failure, as evidenced by diminished force, irregular action, dichrotism, etc. If this estimate be even approximately correct, permit me to register my profound protest. Any drug which increases the force of the heart beat by increasing the load it bears, can not be spoken of as a heart tonic. This is exactly what digitalis does. An arterial constrictor only second perhaps to adrenalin in potency, it has, in my estimation, little or no place in the therapeutics of the hypertrophied, senile heart.

CONCLUSIONS.

(1) While the first requisite for longevity may perhaps be deemed to be an inborn, perhaps inherited quality of tissue structure, wherein one part shall be just as strong as another, like the materials of the famous One Hoss Shay, it is still manifestly possible and sometimes practicable to detect the onset of those pathological processes frequently originating in middle life, and whose development is synonymous with that symptom complex known as old age. Further, when a diagnosis is thus made, it is possible and frequently practicable to institute therapeutic measures that will ensure the prolongation of the individual life.

(2) The study of the processes of disease in the aged, together with their appropriate therapeutics, offers a rich field for individual research, and one that deserves more thorough cultivation. In this connection, our present knowledge of the subject needs thorough revision, amplification, and systematization at competent hands, and to be made more accessible.

(3) It is requisite to the successful study of disease in the aged, and certainly almost essential to the one who undertakes their management, that he be possessed of a peculiar mentality, combining not only a liberal education, but great resourcefulness in tact, patience, and sympathy.

Other things being equal, I am persuaded that practitioners will be most successful with this class of patients whose personal experiences have most thoroughly plumbed the heights and depths of both joy and sorrow, and who can say in his heart "these be mine own people."

SURGICAL ASPECTS IN THE AGED.

OBSERVATIONS OF FOUR YEARS' SERVICE AS ASSISTANT SURGEON TO ILLINOIS SOLDIERS' AND SAILORS' HOME.

GEORGE E. ROSENTHAL, M. D.
QUINCY.

The following paper is based upon fifty operations, occurring during a period of nearly four years. Forty-five of these were under general anesthesia. The mortality rate was 10 per cent.; the average age of the individuals operated upon was 66 years, the youngest was 53 and the oldest was 82. The average age of the fatal cases was 68.

The operations were as follows: Rectal work, 11; amputation of penis for carcinoma, 1; cholecystotomy, 1; leg amputations, 10; thigh amputations, 1; prostatectomies, 5; inguinal herniotomies, 10; femoral herniotomy, 1; external urethrotomy, 1; vasectomy, 2; ligation, external carotid artery, 1; extirpation epithelioma of lip, 4; enucleation of eye for sarcoma, 1; extirpation of tumor of abdominal wall, 1; total, 50.

It would seem, among a population of some 2,000 men, where 70 per cent. of them suffer from prostatic trouble or hernia, that many would seek surgical relief from these ailments. The hernia, however, is always a qualification for pension and they fear if it be repaired it will mean a reduction in the amount of that honorarium, hence they are loath to part with it.

Another deterring factor is their fear of operation, a fear engendered possibly by the memory of the results of operation in that epoch, when asepsis did not grant an eventless convalescence and when the boon of an anesthesia was almost unknown. Moreover, in addition to this constant underlying operative fear, the attitude which these individuals take towards abdominal work is curious. From their viewpoint, entrance into the closed cavity of the abdomen takes on the aspect of a defilement. They will contemplate an inevitable amputation with considerable equinamity but confront the idea of a laparotomy with great repugnance, almost abhorrence. In any event, the indication for operation must be obvious to the lay mind before consent is given.

A study of the table of operations will show an entire absence of laparotomies, except those for hernia and the one gallstone case. It seems that the indication for operation must be visible or palpable, as for instance a hernia or a gangrenous extremity, before operation is accepted. A surgical kidney, a pleural empyema or an enlarged prostate does not furnish this unmistakable evidence of pathological process and so surgical relief is refused.

The contra-indications to operation which we encounter are: 1, the age of the patient; 2, the advanced state of the disease when the patient reaches us, medical treatment having usually extended over a considerable period; 3, advanced arteriosclerosis; 4, chronic bronchitis; 5, kidney impairment or some form of urinary or genito-urinary disease. The advice of the family physician is, as a rule, against operations.

In these cases chloroform is the anesthetic of choice, but full anes-

thesia is sometimes difficult to obtain, because of alcoholic chronic addiction. Nevertheless, serious vicissitudes are rare.

The rectal conditions requiring treatment were, chiefly, hemorrhoids and fistula, with one case of prolapse and one of sigmoid enteroliths. In the latter case, these concretions were smooth, hard and facetted, three in number and together weighed two ounces. They formed a mold of the sigmoid and were removed, per rectum, after some difficulty. The symptom complex was that of a sigmoid carcinoma. A fistula complicated this condition. Among these old men, we notice that the sphincter takes a proportionately longer time to regain its tone after stretching than in younger patients.

The conditions necessitating amputation were: Chronic ulcer, 1; osteomyelitis, 2; frost bite, 1; senile gangrene, 7. In amputations below the knee, we find that the bilateral musculo-cutaneous flap method furnishes the most viable flap, in those with sclerotic arteries. It adds but a few minutes to the time of operation and may be done under local anesthesia. We have never had a slough, when this flap was used, but one cutaneous cuff amputation was followed by this complication. At times, the sclerotic arteries make hemostasis difficult, the ligature breaking through the brittle lime-incrusted arterial walls. This may be obviated by including a sufficient amount of surrounding tissue, excluding any adjacent nerve.

In prostatic cases, we encountered a decided aversion to operation. The part played by the offending gland may be explained at great length, yet consent to operate is withheld until absolute and continued retention exists. We have, however, secured good results by continuous catheter drainage, if the urethra be pervious and be not too tortuous or too difficult of passage. A linen prostatic catheter is sterilized by boiling, is passed into the bladder under aseptic precautions and is secured in place by adhesive straps, to be removed and replaced by another in twenty-four hours. Bladder infection is apt to follow the most painstaking technique.

Of the eleven herniotomies, ten were inguinal and one femoral. Of these, one femoral and five inguinal were strangulated at the time of operation. Of the other cases, one was complicated by cystic degeneration of the testicle. Adhesions to the cord are the rule and are dense and baffling, since the disability has existed for from 20 to 40 years. We notice also, in those having sclerotic arteries, the incidence of more recent hernias, due to the progressive arterial process and the simultaneous retrogressive change in the body musculature. In the same category is the pendulous belly of the aged. The operation of herniotomy is the most satisfactory of any we are called upon to perform and, notwithstanding the age of these men, we have had no recurrence in this series.

The cause of death is almost invariably an acute nephritic process implanted upon a kidney already impaired. This becomes manifest in from one to four weeks after operation, the urine contains albumen in large quantities and many casts, death occurring during uremic coma. The cases resulting fatally were: One prostatectomy after three weeks; one herniotomy after four weeks; another after two weeks; a case of anal

fistula in two weeks and a carotid ligation, followed by death in 48 hours, from aspiration pneumonia.

The dictum of Lawson Tait, to conserve your patient's resistance by finishing your operation in the shortest possible time, is nowhere more pertinent than in these cases.

MANIFESTATIONS OF RHEUMATIC INFECTION IN CHILDREN.*

C. MARTIN WOOD, M. D.
DECATUR.

While in adult life, rheumatism may be generally understood to mean an arthritis, it is quite different in childhood, as the rheumatic infection manifests itself by attacking widely separated structures. In this respect, it resembles most infections in children, which have a tendency to become general instead of remaining localized. Indeed, rheumatism is now looked upon as an infection, having all the characteristics of an infectious disease. F. J. Poynton of London has isolated a diplococcus from the tissues, which, in one case, produced rheumatic fever in a rabbit by inoculation from a culture. While bacteriological investigations are not conclusive as yet, they are suggestive, and the future may demonstrate a specific germ. One who practices among children must be on the lookout for tonsillitis, endocarditis, chorea, torticollis, and erythemas, as well as arthritis as evidences of rheumatic infection.

Dr. R. H. McConnell studied 500 cases of rheumatism in children at the Vanderbilt clinic in New York. These cases showed the following percentages: Tonsillitis, 35 per cent.; endocarditis, 23½ per cent.; articular forms, 23½ per cent.; chorea, 14½ per cent.; muscular rheumatism, 2 per cent.

Subcutaneous nodules and torticollis were present in fractional percentages. Many of these manifestations were complicated with each other but the percentages are given of the most pronounced. While, in many cases, the diagnosis of these conditions is not difficult, in others their recognition requires considerable care. In all cases, a knowledge of the family history and the previous history and predisposition of the patient is of great value.

Tonsillitis, as a rheumatic infection, is recognized by the history and by other manifestations, occurring simultaneously. The pain and aching is greater than we expect to find with the degree of redness and inflammation present. The pain often extends into the muscles of the neck. Salicylates are effective in relieving the symptoms.

Endocarditis may be either primary or secondary. Practically all the primary cases are due to rheumatic infection, whether the other symptoms are pronounced enough to attract attention or not. Nearly all the acute infectious diseases of childhood may produce the secondary forms, as may also septic infection from any source. The history of a preceding

* Read before the Section on Medicine Illinois State Medical Society, at Springfield, May 15-17, 1906.

illness or a suppurative focus would serve to differentiate the secondary forms. The primary or rheumatic form should be looked for carefully in any rheumatic condition and also in a routine examination in any obscure illness. Whenever, in the course of rheumatic arthritis or other manifestation of rheumatism, there is an increase of the fever, with or without increase of the joint symptoms, with a rapid or irregular heart action, with the first sound gradually changing to a murmur, endocarditis should be suspected (Caille).

Arthritis is rare under one year and not common under four or five years of age. In older children, it is more frequent and may be due to several causes. That due to rheumatism must be differentiated from scurvy, gonococcus or suppurative arthritis, tuberculous and syphilitic periostitis. Scurvy is recognized by the general condition of the child, the absence of fever, the condition of the gums and the prompt improvement under proper diet. Gonococcus or suppurative arthritis may be distinguished by the history and by a search for evidences of these diseases in other parts of the body. Tuberculous arthritis is slower in its onset. There may be a history of tuberculosis in the family. The joint surface is not reddened and there is limitation of joint movement, aside from that produced by pain. Syphilitic disease of the joint will be accompanied by other symptoms of the disease and will be relieved by anti-syphilitic treatment.

The frequency in which chorea is a rheumatic manifestation varies greatly, according to different authors. If the term "rheumatism" is used only to include arthritis, the number of cases is small, but if the broader interpretation of rheumatic infection is understood, the number is larger.

Holt believes it to be over 50 per cent., from his observation. In McConnell's list, it is given as 14½ per cent. but, as he remarks, it is significant that he found heart complications in about 70 per cent.

Of the rarer forms, muscular rheumatism has to be distinguished from fatigue and neuralgic pains. The muscular soreness resulting from over-exertion is transient in character, and follows so closely upon the exertion that the cause is apparent. Neuralgic pains are more shifting in character, sharper, and characterized by periods of relief. Torticollis is more rare and may be due to a variety of causes, many of them being reflex, from irritation of the spinal accessory nerve, from bony deformity, Potts' disease, pressure of enlarged glands, malaria, etc. In the form due to rheumatism, the reflex causes mentioned above can be excluded, the affection is more acute and painful, and is best relieved by salicylates. Subcutaneous nodules are said to be rare in this country but would be readily diagnosed if seen. They are small semi-solid bodies appearing along the extensor tendons of the fingers; at the knees or elbows. Erythema nodosum is quite rare, no case being seen in McConnell's 500 cases. I have seen one case, in a girl of 10 years. Notes taken at the time stated that "three days ago she noticed red, raised lumps on the front of both legs below knee and over the shins. Examination shows large flat papules, hyperemic and edematous. Redness extended beyond the raised part,

fading gradually. Temperature 100° F., pulse 124. Under salicylates, she improved and the lesions disappeared in about a week."

The importance of rheumatism, when affecting children, can not be too strongly emphasized. The practitioner should be on his guard and institute proper treatment at the earliest moment. Children should not be dismissed as suffering "only growing pains." The marked tendency of some children to frequent outbreaks in one form or another should be recognized. The treatment of the rheumatic child should aim above all to protect the heart and prevent subsequent valvular disease. The treatment of symptoms as such is of minor importance. Rest in bed is easily secured, during the painful stage of the disease, but should be insisted upon for a much longer period, at least several days after the subsidence of all symptoms. The medicinal treatment is simple and consists of large doses of sodium salicylate and sodium bicarbonate. The alkali I consider of equal importance with sodium salicylate. When given properly diluted, it is rare that they are not well taken. In a child subject to rheumatism, every care should be taken to guard against repeated attacks. Woolen underclothing and good shoes should be insisted upon, and unnecessary exposure prohibited. Exercise and bathing should be carefully regulated, so as to increase the natural powers of resistance. In these children, I always begin the administration of sodium salicylate on the least show of any such illness as a cold, grippe, or sore throat.

115 N. Monroe St.

HEMANGIOMA AND ITS TREATMENT.*

CARL BECK, M.D.

CHICAGO.

Two years ago I read a paper before the Chicago Medical Society and exhibited a few cases treated by a new method, the subcutaneous spiral suture. The results obtained by this method were very superior to the results obtained by any other method I had used before. Since that time I have used the subcutaneous spiral suture on a considerable number of cases and I have satisfied myself that we can obtain almost ideal results with this method. In one of the last numbers of the *Centralblatt für Chirurgie,* Prof. Ali Krogius, of Helsingfors, publishes a new way of treatment of angioma by subcutaneous interlocking sutures, and in his article he quotes my method, saying that it has led to his way of treatment, but that he thought he could obtain equally good results by the interlocking suture. In the same journal Professor Payr, of Graz, publishes his results of treatment of angioma with magnesium tacks. A comparison of the results of these three different methods prompts me to publish my experience with my own, because I think that the subcutaneous spiral suture is superior to the others and because I did not make my method entirely clear in my previous publication.

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*Read before the Surgical Section of the Illinois State Medical Society at Springfield, May 15-17, 1906.

Before I enter into a discussion of the technique, I must state that it is not applicable to every kind of hemangioma. An angioma of blood vessels may appear at different stages of life in different forms. In some of the cases the tumor develops in young children, beginning as a small winemark, which remains stationary and does not enter into the subcutaneous tissues. It is, in reality, not a tumor, but a simple hyperplasia of blood vessels. Some winemarks may grow slightly during the first year of life, or they may begin to grow at the age of puberty to a larger size; they may protrude slightly over the surface of the skin; they may be highly red or of a bluish, discolored tint, but all these varieties are really not tumors, but a hyperplastic condition of the vessels. In these cases the spiral suture is not applicable, but we treat them successfully with electricity. Another kind of angioma also begins as a small winemark, but shortly afterward, or again at the age of puberty, it grows above the level of the skin and into the subcutaneous tissue, forming a real tumor. The vessels themselves are pathologically changed, irregular in shape, disproportionate in size and intercommunicating by loss of the wall between them through pressure atrophy, forming what we call large blood caverns. The larger number of vessels are venous. This kind of tumor becomes very large, grows very rapidly and shows a tendency to malignancy. It destroys, by its pressure and growth, tissue as hard as bone or cartilage. This rapid growth is especially noticeable in infants and young children, and within a few months or even a few weeks a tumor of considerable size may grow out from a small winemark. They appear on any part of the body, but more frequently on the face, cheeks, within the mouth, on the tongue, etc. This tumor is pre-eminently fit for treatment with my method.

A third kind of angioma is one in which the arterial blood vessels prevail. This growth is characterized by pulsation, erectility, etc. It is also amenable to treatment with my method, but with a modification, namely, the preceding ligature of large supplying vessels, for instance, the carotid artery or the obturator artery.

A fourth class of angioma is one which, in reality, is a mixed tumor, involving, besides the blood vessels, a large amount of fat, connective or glandular tissue. In the treatment of these cases a great deal of surgical ingenuity and tact is required in order to obtain permanent results.

I admit that, in some instances, Krogius' method may be preferable, perhaps in the case of erectile angioma, but it would have to be tested first on a few cases.

My method of treatment is by no means entirely original in surgery, as the ligature had been used for the treatment of tumors a hundred years ago. One feature of it, however, is original, the removal of a tumor without much disfigurement. This is brought about by the use of a physiologic phenomenon in the treatment. We have to operate in many sittings on the same individual and exterminate the tumor mass gradually by diminishing its volume, and, while we are doing this, we enlarge the adjoining normal skin to the same extent, so that when the tumor has reached its smallest size the normal skin has grown to its largest extent,

and when the tumor has been reduced to a fibrous lump of scar tissue the skin is normal around the small lump, and when extirpated leaves hardly any scar. The method consists practically in this: Before any suture is employed we study in which direction the scar afterwards will run, as the constriction of the tumor must be done in such a manner that no ectropion or disfigurement by scar may occur. This will be better understood by an illustration. Suppose we have an individual case with an angioma of large size underneath the eyelid. The ultimate scar should run in the direction of the nasolabial fold. We have to enlarge the skin from side to side in order to prevent an eversion of the eyelid. This side-to-side traction must also be the traction of the spiral suture. The spiral suture must, therefore, pass from the nose toward the ear and from the ear toward the nose and back and forth in this direction. If we should, by mistake, make this spiral suture from the eyelid to lip and back and forth, we would not only not constrict the tissue, but we would draw up the lip and pull down the eyelid, leaving a very disagreeable condition, which we could not remedy very well afterward. Besides, the skin would not grow in that direction, up and down, because there is no tension between those two points. If we want to make an experiment in order to show that skin can grow between two points, we can satisfy ourselves easily in this manner. If we take a skin under fair tension, for instance, on the arm, and make two scars about five inches apart and then raise a fold between those two scars by a subcutaneous spiral suture, we may bring the distance of five inches down to three inches. If this fold remains persistent, some time later the skin will be farther apart, perhaps four inches, and, later on, about five inches. This shows clearly that the skin has grown fully two inches between the two scars. If we release the fold, by cutting the adherent tissue, which has been brought together by the spiral suture, the skin will atrophy to the original size so that the scars will be again, after a time, only five inches apart. We can notice such an enlargement of skin in pendulous tumors, which cause quite an enlargement of the skin through a traction produced by their weight. Upon removal of those tumors, the skin which has been so abundant gradually atrophies, through non-activity or non-irritation, to its original size. In my method of the subcutaneous spiral suture, I make use of this physiological growth of skin.

The spiral suture is commenced by passing the thread from one point of the periphery of the tumor underneath its base to the opposite side, then back and a little sideways under the surface of the skin, so that the needle comes out slightly to the side of the first puncture. This is drawn fairly tight and the thread is passed again through the same stitch, pole underneath the base, coming out at the opposite border somewhat to the side of the second puncture, going back again underneath the surface and so on until the whole tumor is enveloped in a spiral suture between the two most distant points. The suture is drawn as tight as possible, and, when the spiral is finished, the thread is carried back to the first point of entrance and the two ends of the thread are tied in such a manner that the knot is buried somewhat by the tension. The

thread is out of sight, as it is entirely buried. Since it does not cut off the circulation entirely, necrosis of the tumor or of the skin above does not occur. The points where the needle has pierced the skin are covered with collodion. Occasional measurements, between the sittings, have shown me that from suture to suture the tumor was diminishing in size and the skin increased in size in the same measure. Ultimately the tumor has been reduced to a fibrous nodule, which does not yield to any suture. Electrolysis might then be used to destroy it. In some cases, at least, this seemed feasible, but excision was much more simple and, with very fine stitching of the borders, the scar is noticed very little.

I will not go into the details of the history of the cases, as they are very much alike and one history reads very much like the others, but will take only one case as an example, the child of Mrs. B., of Seattle, 8 months old. When the child was born, it was perfectly normal, the face being without a blemish. Two weeks later a little spot was noticed underneath the outer corner of the right lower lid. This little spot increased to the size of a dime, when it became slightly prominent and of a grayish discoloration. The doctor who had the case in charge consulted one of the surgeons of Seattle, who, after two weeks of observation, advised an operation, because the tumor grew to three times its size in those two weeks. He made an eliptic excision and stitched the two borders of the skin. Primary union took place. Two weeks later, however, the scar spread slightly and small veins appeared within it. A bulging of the scar and a growth on the side a few days afterward alarmed the parents and they came to Chicago, where their parents resided. The grandparents of the child were neighbors of a family in which I had operated for an angioma of the nose, and this family referred the people to me. When I saw the child it was 8 months old. The tumor filled the larger part of the cheek, with a scar running on the surface of it, and was slightly pulsating. It was one of the angiomas which I have placed in the second class of the above division. I began with a spiral suture of the tumor. Within three months I made eleven subcutaneous sutures, with the result that a nodule of one-half inch in size of hard connective tissue remained. After excision of the nodule and fine coaptation of the borders, the result was ideal. One and one-half years have passed since the operation and there is hardly a perceptible scar under the eye.

Taking into consideration the simplicity of technique, the absolute safety of the method and the possibility of ambulatory operation, it seems to me that the subcutaneous ligature is preferable to the otherwise excellent method of Payr with magnesium tacks. The introduction of magnesium is something that needs watching. It requires the patient to stay in a hospital a long time, which is quite difficult in the case of infants. The other methods which have been advocated and which certainly give results, like the introduction of hot water, seem to me too risky in cases of tumors of any extent. I have now observed a sufficient number of cases to justify me in saying that the subcutaneous ligature in spiral shape is the best and least inconvenient method of treatment.

In cases of very large arterial, venous or mixed tumors, extirpation will have to be considered. During the last year I have had occasion to observe one of those monstrous angiomata in a young married lady, which had existed perhaps since birth. It had grown to an enormous size during childbirth. It was the second case of this kind I had seen in my practice. The first I described fourteen years ago, with Dr. Wagner, in the *Chicago Medical Recorder*. During confinement the tumor had burst, and the hemorrhage, which almost exsanguinated the patient, could not be stopped by packing, ligature or any other method except by continuous sutures of the tissues in different directions. This last case of angioma, observed during the last year, was of the size of a man's head, so that when the lady was standing up a large pulsating tumor hung down from one of her buttocks. It caused a great deal of pain. I concluded to remove the tumor. The lady entered the Michael Reese Hospital and, with the kind assistance and help of Dr. Lester Frankenthal, in whose care the lady was previous to and after confinement, I operated on her about a year ago. I was surprised that the tumor had not interfered with the confinement, and I think that it must have been extremely difficult and taxed the skill of the obstetrician to prevent a hemorhage during confinement, which certainly would have proved fatal. Our operation consisted in a flap incision, extending from the coccyx to the outer crista ilei. After the flap had been dissected back I ligated every bleeding vessel and worked my way along the angioma toward the pedicle. Notwithstanding forceps and an enormous number of ligatures, constant bleeding could not be prevented, so that when I had worked my way toward the pedicle in the region of the pyriform muscles in the incisura ischiadica, I had to place a ligature *en masse*.

The patient was returned to bed almost pulseless, and only through the very energetic and careful treatment of Dr. Frankenthal were we able to resuscitate her sufficiently to obtain a pulse. During the first twenty-four hours, her condition was critical. The anemia was extreme, but during the next eight days she gained rapidly, when, during a change of dressings, a profuse secondary hemorrhage took place. An interne was called and within a few minutes such a hemorrhage had taken place that the lady was brought again nearly to a point of danger. The interne packed the wound through the opening left for a drainage tube and stopped the bleeding. At the end of two weeks another secondary hemorrhage took place, which necessitated the rapid reopening of the cavity and suturing of one of the bleeding arteries. Then the wound was treated openly and ultimately healed. This case illustrates the treatment of the third and fourth class of angioma—the angioma arteriale.

DISCUSSION.

Dr. A. E. Halstead, of Chicago:—In regard to the treatment advocated by Dr. Beck, I have had cases of angioma of the cheek, some of which were treated with spiral suture. The patients made a good recovery without deformity. I wish to mention a class of angiomata that are not to be treated by this method, and I refer to those angiomata that occur on the inside of the cheek. I recall one case I had recently, where attempts were made in the case of a small girl to excise the angi-

oma, which occupied the inner side of the cheek. The tumor was the size of a man's fist and protruded from the mouth. It was partly removed by a surgeon a year ago, but recurred and grew larger than it was at first. It seemed impossible to excise the tumor without greatly disfiguring the face, and it was impracticable to use the spiral suture because of the situation. Accordingly I adopted the plan recommended by Wyeth, namely, the injection of boiling water, and had a very gratifying result. The patient's face became swollen after the injection of the water, but the swelling subsided inside of a week and the patient left the hospital at the end of two weeks without any trace of the angioma. The cicatrix was not larger than the end of the thumb. The tumor originally was larger than a fist. For these cases, where the ligature is not applicable, I can strongly recommend this treatment of Wyeth's. The technic is simple. You can use any kind of syringe. The syringe I used was one that is employed for antitoxin, and it is only necessary to keep the water boiling and inject it into the tumor. Following the operation there is no pain or sloughing and the cure is complete. When the tumor is venous and communicates with a large vein, preliminary ligation of the vein will prevent a possible embolism.

Dr. Charles J. Drueck, of Chicago:—Dr. Beck, in closing his remarks, stated that the thread has disappeared. I was wondering whether he used silk and had to cut it out afterward or whether he used catgut. I would like to know which material he used.

Dr. E. Mammen, of Bloomington:—I would like to ask Dr. Beck what kind of material he uses for that purpose.

Dr. J. F. Percy, of Galesburg:—I am interested in this paper and particularly in the first case Dr. Beck has mentioned. We all know what distressing cases they are. When a young woman wants a mark of the greater degree of angioma removed from her face we are usually up against a very difficult proposition in attempting to select the best method.

I hope I am not out of order in mentioning the fact that I have used, in a number of cases, the method whereby the blood is coagulated in the capillaries by spraying superheated air over the affected surface. The apparatus used is that of Holinger, with which he first began the treatment of lupus. This is merely a Bunsen burner with a coiled tube, at one end of which is a nozzle and at the other a rubber bulb, such as you use on the Paquelin cautery. Under chloroform anesthesia superheated air is sprayed over the affected area. It produces a burn of the first degree. You keep applying it until the tissues acquire a sickly yellow hue. I have had some gratifying results by this method, but no complete cures, mainly because the patients are so improved that they think a cure has been effected. They tell me they notice that people do not look at them any more on the street, and that is such a relief to them that they get tired of repeated anesthesias. I have had one case that I have anesthetized four times, and it seems in many of the cases the capillaries get larger as we go deeper in these so-called cases of port-wine marks.

I am gratified to know of this method in this second class of cases.

Dr. Beck (closing the discussion):—The suture material used was catgut. At first we employed silver wire, but afterward it was found that catgut was the best material, and it disappeared within ten or twelve days entirely from the tumor and left the tumor in a retracted condition.

So far as the first class of tumors referred to by Dr. Percy is concerned, I have treated them with the hot-air treatment, and it produces a surface-burning and cicatrization which is not very regular, and on top of the cicatrix there appear again small veins. We have a different apparatus, consisting of a syringe in which there is an electric bulb, which gives usually good results; you have got to have quite a high temperature to compress the air passing over the electric bulb. The results are not as good as with the treatment by electrolysis. The latter has the disadvantage, in that you have to anesthetize a patient ten or twenty times to remove the small marks. In angioma of the nose these methods have given variable results, with a small amount of scar and very little disfigurement. The

treatment is simple. One electrode is applied to the cheek and the other to the tumor, and then with a little gas foaming up it shows that we have enough destruction of the tissue. And this I repeat at different points, say a distance of from one-tenth to one-quarter of an inch, and at different times until we have destroyed the larger vessels.

The treatment with hot water, referred to by Dr. Halstead, has also proven successful, but not as successful as in the case cited by Dr. Halstead of surface angioma, because I have had extensive gangrene following it. I do not know why it should have occurred in this case. I had another case of surface angioma in which there was produced gangrene in the mouth. The result was very good, but there was quite a retraction, so that the movement of the jaw was interfered with.

ILLINOIS MEDICAL JOURNAL

THE OFFICIAL ORGAN OF THE ILLINOIS STATE MEDICAL SOCIETY.

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OCTOBER, 1906.

ILLINOIS MEDICAL COLLEGES AND THEIR STANDING.

The following correspondence, published in *The Journal* of the American Medical Association for September 29, is of so much interest to the profession in the state, and especially to the members of our State and County Societies, that we reprint Dr. Egan's letter in full, as well as the comments of the editor of *The Journal*. In the interests of medical education and the advancement of the profession, no efforts should be spared to insure a correct understanding of the tables and reports of the Council on Medical Education and the inference to be drawn from them, regarding medical colleges in Illinois.

STATE BOARD RESULTS IN THE EDUCATIONAL NUMBER.

SPRINGFIELD, ILL., Sept. 17, 1906.

To the Editor:—Please allow me space to dwell further on the erroneous conceptions which may be gained by the casual reader through a misinterpretation of the significance of the data in the educational number of *The Journal*, August 25.

I have now before me a printed postal card which I believe has been sent broadcast, bearing the bold-faced caption, "Failures of Chicago Graduates," and giving a list of the different medical colleges of Chicago with the percentages of failures of their students before the "different state medical examining boards throughout the United States during the year 1905." *The Journal*, Aug. 25, 1906, is quoted as the authority for the figures.

This card shows the National Medical University as the Chicago medical college established over five years having the smallest percentage of failures, with

the Northwestern University Medical School and Rush Medical College in the second and third place, respectively. The reader, conversant with the standing of the two older colleges, among the medical institutions of the state, is at first skeptical, but on referring to your educational number (Table A) he finds that the author of this card is fully supported by your figures, and was guilty of no misrepresentation when he quoted *The Journal* as his authority.

This incident illustrates forcefully the danger of the publication of deductions drawn from data whose figures are not uniform in significance. To the vast majority of the readers of *The Journal* to whose attention this card may come, the statements on the card are fully substantiated by reference to your Table A, and they will not take the trouble to go further to ascertain the exact facts. We have here, in short, a very widely promulgated misconception, for which *The Journal* is unfortunately made to stand sponsor.

It is true, as you have said, that your readers were cautioned, in the editorial accompanying these data, to consider the conditions surrounding their origin, but it is my impression that a very large percentage of those who glanced over the deductions of your tables paid little or no attention to their discussion and explanation. The average reader is prone to accept conclusions, especially when based on figures, with little concern as to their source.

On an analysis of your figures we find that the National Medical University, the school credited with the highest standing, had 24 applicants who appeared before but 7 examining boards. One of these applicants failed to pass an examination. Northwestern University Medical School had 191 applicants before 27 boards, and 10 of these failed to pass an examination. Rush Medical College had 254 applicants before 30 boards, and 21 failed to pass an examination.

Of the failures of Northwestern 6 were before boards which rejected from 25 to 50 per cent. of all applicants from other states. Of the failures of Rush Medical College 10 were before boards rejecting 25 per cent. or more of applicants from other states. The National had but 1 graduate before any board which rejected more than 25 per cent. of applicants, and that one failed.

As I said in my communication in *The Journal*, September 15, "we might as well eliminate from our data the showings of colleges whose graduates have appeared before only a small number of boards, as these showings indicate little as to the ability of the graduates of such schools to pass the average state board examination."

It is obvious that percentages drawn from the successes and failures of the National Medical University and the other two institutions are in no way comparable. I am not prepared to say what success the graduates of the school given highest place would have attained before a larger number of boards, but for 1905 the percentage is based on results before an insufficient number to give an adequate idea of the standing of any school. Still *The Journal*, through the publication of these deductions and percentages, is compelled to stand sponsor for statements which are very widely promulgated and used to influence the young men of the country in the selection of the place for their medical education.

JAMES A. EGAN,
Secretary, Illinois State Board of Health.

EDITOR'S NOTE: Our correspondent refers to a postal card which, although anonymous, leaves little doubt as to its source. It is to be noted how skilfully its author, by the use of the words "colleges established five years or more," omitted one college, an evening school, which happened to have a still lower percentage, by reason of having had only one graduate examined outside of Illinois, and he failing. A college that had received, in an exceptional year, and often from a combination of circumstances, an unusually low percentage of failures could hardly be blamed for taking advantage of it, since another opportunity might never occur. That it should send out anonymous statements, for the purpose of conveying a wrong impression, as the cards referred to doubtless were sent out, only adds to the suspicion with which at least one Chicago college is regarded. This suspicion our correspondent seems to share. If the suspicion is not well founded, why is it not

as fair for one college to be shown up as having received a low percentage as another? As to the figures being insufficient, the college had 29 graduates, 24 of whom took the examination. This would seem to be a large enough proportion for a fair basis for figuring the percentage.

The statistics published are uniform except in degree in which they never could be uniform, and therefore could never be strictly "uniform in significance." Because a casual reader may form a wrong conclusion or because a college so inclined may publish half-truths for selfish purposes, are not good reasons for withholding from publication statistics which are of absolute value to the careful reader.

While there are bound to be differences between the percentages of the same college from year to year, each year hereafter will furnish larger totals from which to make deductions and then the deductions, of course, will have added value. For the past three years the figures for Chicago colleges are as shown in the accompanying table. It will be noted that the college which appears first on the anonymous postal card takes a back seat at this table.

TABLE SHOWING RESULTS OBTAINED BY GRADUATES OF CHICAGO COLLEGES IN EXAMINATIONS BY STATE EXAMINING BOARDS HELD DURING THE YEARS 1903, 1904 AND 1905.

Chicago Colleges.	Examined in all States.				Examined in Illinois.				Examined in Other States.				No. of States Examining.
	Total.	Passed.	Failed.	Per Cent. Failed.	Total.	Passed.	Failed.	Per Cent. Failed.	Total.	Passed.	Failed.	Per Cent. Failed.	
Northwestern Univ. Med. School...	477	460	17	3.6	217	217	0	0.	260	243	17	6.5	29
Rush Medical College	784	732	52	6.6	359	357	2	0.6	425	375	50	11.8	29
Coll. of Physicians and Surgeons..	790	729	61	7.7	450	447	3	0.7	340	282	58	17.1	31
Hering Medical College	55	50	5	9.0	30	29	1	3.3	25	21	4	16.0	17
American Med. Missionary Colleg .	48	43	5	10.4	11	11	0	0.	37	32	5	13.5	20
Hahnemann Medical College	204	182	22	10.7	132	129	3	2.3	72	53	19	26.4	23
American Coll. of Med. and Surg..	77	65	12	15.6	60	55	5	8.3	17	10	7	41.2	9
Jenner Med. Coll.	54	44	10	18.5	49	42	7	14.3	5	2	3	60.0	5
Bennett Medical College	77	62	15	19.5	45	43	2	4.4	32	19	13	40.6	17
National Med. University	65	51	14	21.5	49	39	10	20.4	16	12	4	25.0	9
Illinois Med. College	164	121	43	26.2	69	61	8	11.6	95	60	35	36.8	32
College of Medicine and Surgery...	63	39	24	38.1	52	36	16	30.8	11	3	8	72.7	8
Dearborn Med. Coll.*	47	46	1	3.4	46	46	0	0.	1	0	1	100.0	2
Totals.....	2905	2624	281	9.7	1569	1512	57	3.7	1336	1112	224	16.7	

* Unclassified, since figures are for two years only, and all but one were examined in Illinois.

THE PASSING OF DOWIE.

John Alexander Dowie, some time First Apostle of Zion and the most successful religious fraud of the present time, has gone the way of all such adventurers and has been forced to leave the organization which he so skilfully built up during the past fifteen years. One of Dowie's strong points was his abuse of the medical profession, and yet, according to recent statements, he has been obliged to avail himself of medical aid in his efforts to keep alive. It seems probable that the city which Dowie founded will rapidly disintegrate and that another ten years will find Dowie simply a reminiscence. In the mean time, we wonder what form of religious fraud will next appear.

STATE HOME FOR EPILEPTICS.

According to a recent report, the State Board of Public Charities is about to undertake the important duty of providing a suitable home for the numerous epileptics of the State of Illinois. At the October meeting, the matter of converting the Soldiers' Orphan Home at Normal into a state hospital for epileptics will be considered and a bill prepared for the introduction to the Legislature next January.

There are only about three hundred orphans at the institution and the plan is to transfer them to the Soldiers' Home at Quincy. The probability is that if this is done the number of soldiers' orphans seeking assistance from the state will rapidly diminish. The Civil War having ended more than forty years ago and all dependent soldiers and soldiers' orphans having been amply provided for by the government, it would seem that there is little excuse for the further continuance of a soldiers' orphans' home. Certainly there is no such necessity for an institution of this sort as there is for a proper provision of the one hundred epileptics in the state.

The State Board of Charities having taken the initiative in this matter, it is to be hoped that all physicians will lend their influence to complete the plan as outlined, especially since all children at Normal can be as well cared for at Quincy.

A MODEL DOCTOR'S WIFE.

Last month there died, in a distant Ohio city, a woman of the past generation, whose virtues should be told as a model, for the present and future generations, of that heroic class known as doctors' wives. This lady, Mrs. Dr. G. W. Fringer, formerly of Pana, Illinois, represents an ideal of what a doctor's wife should be. Her self-sacrifice is more to be admired when it is recalled that she lived and performed her duty at a time when furnaces and steam plants to heat houses during the middle hours of the night were unknown and there were no gas or gasoline stoves to lighten the burden of her labors.

Not only did Mrs. Fringer provide for her husband during the day, but it is said that he never was called out at night that she did not arise also and see that he was properly clothed and equipped for the trip, and many times she accompanied him with a lantern to lighten the dark road and give an encouraging word to cheer him on his way. If she did not go with him, she awaited his return and prepared a warm lunch for his refreshment.

The devotion of the doctor and his wife to each other, as may be imagined, was ideal and a great pleasure to those who had the privilege of entering their household. A few years since Dr. Fringer was called home, where he, no doubt, awaits the coming of the faithful companion of his labors on earth.

COUNTY AND DISTRICT SOCIETIES

ADAMS COUNTY.

The regular meeting of the Adams County Medical Society was held Monday, September 10, at the Elks Club, in Quincy, with President Grimes in the chair. Those present were Drs. Ashton, Beirne, Besker, Bates, Christie, Ericson, Grimes, Hart, Hinton, Kosh, Knox, Montgomery, Nickerson, Pittman, Robbins, Rice, Rosenthal, Schullian, Shango, J. B., Shango, K., Walker and Williams, W. W. The applications of R. T. Hinton, O. F. Schullian and A. S. Stocks were read. The following were elected to membership: W. A. Garner, Clayton, Thos. R. Whray, Golden, A. E. Kidd and J. F. Roach of Quincy.

The subject for discussion was summer diarrheas in adults and children. This was opened by Dr. Knox and participated in by Drs. Becker, Beirne, Hinton, Nickerson, Montgomery, Schullian and Williams. It was the general opinion that, after an initial calomel or castor-oil purge, the treatment should consist of the restriction or withdrawal of food, giving of boiled water, and irrigations of the large bowel with normal saline, at a high temperature. In cases of prostration, hypodermoclysis should be given. The astringents, zinc sulphocarbolate, the salts of bismuth, tanalbin, alone or in combination with minute doses of opium were advocated. In the cases of cholera morbus, which forms about 2 per cent. of the summer diarrheas, the first indication is to overcome the collapse. Dr. Hinton spoke of the remarkable efficacy of Heubner's method of infant feeding in the diarrheas of infants, after the initial calomel purge and hypodermoclysis. Dr. Ashton related some of the events of interest at the Toronto meeting of the British Medical Association, which he attended. The attendance was 2,200; of this number 50 per cent. were Canadians and American visitors. The recognition accorded Dr. W. J. Mayo was most complimentary to him and to the American profession. His paper was on the subject of "Duodenal Ulcer," and he was given additional time for its reading by the chairman, Sir Hester Cameron, on account of the excellence of his paper and because of the general interest manifested. Adjourned.

GEO. E. ROSENTHAL, *Secretary.*

COOK COUNTY.

CHICAGO OPHTHALMOLOGICAL SOCIETY.

Meeting, March 13, 1906.

DR. GEORGE F. FISKE, President.

PECULIAR EXPERIENCE WITH DIONIN.

Dr. Suker reported the following case: As the result of the explosion of a percussion cap, a young man, aged 21, developed a sero-plastic iridocyclitis. In order to get as much dilatation as possible, dionin in powder form was used. Contrary to expectation an excessive myosis developed. The pupillary opening was scarcely visible, and, even under the use of atropin, it was several hours before a pinhole dilatation could be secured. Two days later, after all irritation had subsided, iridectomy was performed, and it was found that even in this short time, attachment of the posterior surface of the iris to the anterior capsule of the lens had taken place. No similar case is recorded in the literature. The question arises whether the myosis was the result of an idiosyncracy on the part of the patient, or due to the impurities in the preparation of the drug from morphin. To determine this the same powder was used on another patient who had iridocyclitis, without producing any unusual effect.

Dr. Faith had seen this case with Dr. Suker, and also another case of mild uveitis, no iritis, in which the dionin caused a marked myosis; the patient was under atropin and the pupils were widely dilated. The physician who had the case under his care tried it again with the same result.

In reply to Dr. Pusey's question Dr. Suker stated that the tension was not altered, either before or after the use of the dionin.

Dr. Dodd had seen slight myosis in several cases following the use of dionin.

Dr. Loring stated that he had inadvertently put one drop of 5 per cent. solution of dionin in each eye of a 12-year-old boy, whose pupils were fully dilated from several days' use of atropin. Fifteen minutes later the pupils were contracted to about 3 mm., and so remained for about an hour, when they began to dilate slowly; in half an hour more they had attained their normal size. (The boy had a marked central chorioiditis and nystagmus, with a high refractive error.) He used the same solution in two or three other cases, with the usual result. The vision was not altered sufficiently to produce a chemosis.

Dr. Colburn cited two cases under observation; in the one he used dionin with the usual result, but in the other no chemosis followed. Thinking that he might have been careless in instilling the drops, he repeated the procedure, but still no effect was observed. In both cases, however, he noticed that in from twenty minutes to half an hour after the use of the dionin, the pupils were contracted. No atropin was used in either of the cases.

In closing, Dr. Suker stated his preference for dionin in powder form and cautioned against impurities which the drugs may contain.

LOCAL DEPLETION IN THE TREATMENT OF THE TRACHOMATOUS CONJUNCTIVA.

Dr. Nils Remmen believes that the roller forceps and strong astringents only irritate a part, which should be left alone; one should evacuate the follicle and deplete the tissue with the least possible increase of blood supply to the conjunctiva. With this in view he uses the sharpened edge of a toothpick to scrape open and evacuate the follicle and exsanguinate the diseased area and then applies bichlorid of mercury, 1/5,000, to the exposed surface. Prompt cure has followed in a number of cases which resisted the more violent form of treatment.

DISCUSSION.

Dr. Fiske stated that he has not used silver or copper in the treatment of trachoma for fifteen years. Instead of the toothpick he uses a cataract knife to scarify. His results have been much better than with the use of the blue-stone.

Dr. Young said that he did not think that strong astringents could be discarded entirely; in fact, he thought that sometimes they are not used in sufficient strength.

DIABETIC RETINITIS.

Dr. W. E. Gamble exhibited and reported the following case: F. P., age 35, R. and L. V. 3/200; examination of the fundus revealed white, highly refractile, widely scattered, punctate areas of degeneration, with a few almost imperceptible hemorrhages throughout the posterior segment of the retina of each eye; the retina between the affected areas was perfectly clear. No history or evidence of syphilis could be found. The urinalysis showed 3,750 c.c. to have been passed in twenty-four hours, a specific gravity of 1,031 and 1.67 per cent. of sugar; no albumen or casts were found, subsequent urinalyses showed 2 and 3 per cent. of sugar respectively. Despite the increase in the amount of sugar the vision had improved to 12/200 after six weeks of observation; the areas in the retina have improved somewhat.

The case falls into Class 1 of Hirschberg's classification, namely, central punctate diabetic retinitis, rather than into Class 2, in which only hemorrhages are present. Other things being equal, the prognosis should be more grave in the hemorrhagic type than in the form without hemorrhages. The areas in the degeneration in the author's case differ in no way from those found in albuminuric retinitis outside the macula.

The prognostic significance of retinitis in diabetes has recently been discussed by Nettleship. He bases his conclusions on 48 cases. The patients ranged from 41 to 79 years; 38 were known to have died—9 in the first year, 11 in the second, 18 between 2 and 8 years after the discovery of the retinitis; 10 were known to have been living 2 years after such discovery, 6 at periods of from 6 to 10 years after. The prognosis as to duration of life, therefore, differs widely from that of albuminuric retinitis, since two-thirds of the patients were living two years after the retinal lesion was observed.

DISCUSSION.

Dr. Dodd referred to three cases of diabetic retinitis in his own experience, one of which returned after four years with improved general health and vision, although the spots in the retina were still present.

Dr. Suker asked whether the vascular tension had been taken. Diabetes is a toxemia, and all toxemias increase the blood pressure, either locally or generally. Prior to the specific lesions appearing in the retina there is always to be seen a retinal haze, the result of the increased peripheral tension; the higher the blood pressure the more apt are hemorrhages to occur, and vice versa. The acme of the fundus findings is in consonance with the tension.

If the tension is low the condition is apt to be of the inflammatory type; if the tension is high, hemorrhages will in all likelihood take place. He has examined quite a few of these cases with Dr. Elliott and in a number of instances has been able to state the condition of the patient from a study of the fundus alone. In all these cases either sugar or albumen was subsequently found. He considers the question of the vascular tension a most important one in the prognosis of the case and urges that it be determined in every instance.

Dr. E. V. L. Brown referred to a case which is an exception to these rules laid down by Dr. Suker. The case is one of bilateral detachment of the retina in a nephritic with thrombosis of the choroidal vessels. The blood presure is 210, and yet there are but three hemorrhages in the two fundi.

Dr. Wilder also cited a case of retinitis in chronic nephritis with arteriosclerosis, in which there are but two or three hemorrhages, although the blood pressure is 240. There is in this case a marked papillitis, such as is often seen in brain tumor and gumma; the patient is totally blind, except for the central vision which often persists to the end.

Dr. Suker said that active congestion is more apt to be found with high tension, and passive congestion with low tension; hemorrhages may occur in either condition, but they are more likely to occur with high tension. The high tension in arteriosclerosis necessarily leads to nephritis. If there is a cardiac lesion, a valvular lesion associated with the high tension compensation will minimize the congestion.

Dr. Gamble stated that the blood pressure was not taken in his case; the arteries were not hard nor did they indicate a high vascular tension, and the absence of haziness between the spots in the retina would speak against it.

HYALIN DEGENERATION OF THE CONJUNCTIVA.

Dr. Wilder presented a patient, a man, aged 50, who has had sore eyes for six years or more. The trouble began in the right eye, with a growth at the inner canthus. Two years ago a similar growth appeared in the left eye, and now it has formed the condition of symblepharon. The growth has almost covered the cornea of the right eye so that vision is very much reduced.

The growth looks like an epithelioma, and palpation would tend to confirm this impression. A microscopic section was made of a small portion of the tumor. There is beneath the conjunctival layer of the epithelium a proliferation of the lymph vessels, and in the connective tissue reticulum there are seen lymph vessels the walls of which are thickened and have undergone hyaline degeneration. In all probability the case is one of hyaline degeneration of the conjunctiva, a condition which frequently follows trachoma.

He has seen two other cases of hyaline degeneration coming on after trachoma. Both patients were Jews from eastern Europe. The treatment is operative. It will be necessary to excise the mass and do a plastic operation on the lid.

Dr. Dodd stated that he had a case which seemed to be very similar to Dr. Wilder's. The entire cornea was covered with the growth. The conjunctiva was very much thickened on all sides in both the upper and lower cul-de-sac. The eyeball was fixed. He removed the growth as carefully as possible and made a large skin graft so as to form a new cul-de-sac both above and below. Finally he was forced to operate again at the inner canthus. What was left of the conjunctiva began to proliferate, and gave much trouble. The cornea became opaque, and it is now thickened and steamy, and the patient complains continually of the irritation. The eyeball has retained good movement, however.

Dr. E. V. L. Brown exhibited cases of keratitis profunda and iritis gonorrhœica.

Meeting, April 10, 1906.

The regular monthly meeting of the society was held in Booth Hall, Northwester University Building, Tuesday evening, April 10, 1906, with the president, Dr. George F. Fiske, in the chair.

CARDIOTHYROID EXOPHTHALMUS.

Dr. G. F. Suker: This man, negro, aged 35, has an excessive myopia, and a very wide pupillary distance, therefore modifying markedly the Moebius sign, which I think is purely a mechanical one that has nothing to do with the disease itself. The von Graefe sign is not perfect; he has, however, a uniform lagging of the lid. Last October, when first seen, he complained of blurred vision in the right eye and failing vision in the left eye. On examination I found that he had rather a profuse intraocular hemorrhage, which was absorbed after a time, although the fundus of the left eye remained indistinct and clouded for some time. His pulse was 140, and arterial tension 150, 160, 170 and at one time 200. The patient was put to bed and given thyroidectin under which both his general condition and vision rapidly improved. His vision now is 20/50 and the fundus practically clear; his pulse is 110, and the vascular tension 120 to 130. I begin to look on this hemorrhage as the result of high arterial tension. As the circulatory stagnation and peripheral resistance diminished, the fundi cleared up and he improved generally. Dr. Elliott has collected fifty or sixty of these cases, in all of which the tension was above 130, the maximum being something over 200. All the patients had eye symptoms, and all had blurred vision. My patient, after he got out of bed, went back to work. His pulse and tension immediately went up and his vision began to drop. He was put to bed and again he immediately improved. Here, as in the case I referred to at the last meeting, the arterial tension gives the key to the situation. The retinal arteries are among the first to suffer from high tension because they are end arteries.

A NEW TRANSILLUMINATING APPARATUS.

Dr. H. V. Würdemann:—The transilluminator devised is a modification of the DeZeng ophthalmoscope. The entire instrument is not much larger than a fountain pen, and may me handled in the same manner. The small electric globe in the ophthalmoscope is replaced by another of the same size on the end of which there is a lens. The cone-shaped screw cap of the instrument contains a glass rod about three-fourths of an inch in length, which comes in contact with the end of the lens. Nearly all the light is thus focused in the glass rod, and is brought out through it to the tip of the instrument and from there a beam of light is projected through the eye or the structures to be illuminated.

The instrument is also efficient for illuminating the frontal and ethmoidal sinuses. The cost of the instrument is about $5. It is of great service in differentiating intraocular tumor from pus in the eye and other conditions that may be mistaken for tumor.

HYPERPHORIA.

Dr. J. E. Colburn reported a case of hyperphoria of 9 per cent., with limitation of upward motion of the fellow eye amounting to 10 per cent.; an advance-

ment of the superior rectus of the first eye and two subsequent tenotomies of the corresponding muscle of the fellow eye gave only temporary relief. Exploration of the inferior rectus tendon of the second eye then revealed a broad adventitious band, 2/3 the size of the head of the tendon, limiting the motion of the eye upward. Eighteen months have elapsed since the excision of this band and orthophoria has been secured, along with the cessation of all the symptoms. Following the first operation astigmatism of 5 D. axis 90 developed; this has gradually decreased since the last operation and is now only a little more than 3 D.

DISCUSSION.

Dr. H. V. Würdemann:—The experience is so unusual that I can scarcely discuss the operation or the condition of the muscles. It is certain that, after an advancement, we do find some astigmatism, but I have never known the condition to become permanent after an operation on the ocular muscles.

Dr. J. B. Loring:—The tripometer should have been of value in determining the ability of the eye to rotate in this case; upward rotation would have been defective.

Dr. Colburn, closing the discussion:—The astigmatism in this case increased each time I advanced the muscle, and up to about the same degree. It then slowly decreased, and increased again with the next operation, and so on. After the last operation the astigmatism dropped 2.5 D. almost immediately.

As to a permanent change in the astigmatism following operations on the muscles, I have frequently seen astigmatisms increased and decreased following operations for tropias and phorias, changing up to 1 D. or 1.5 D. I stated in my paper that the rotation upward was 10 degrees less than the rotation of the fellow eye: I have seen that occur in a weak or insufficient muscle.

THE DIPLOBACILLUS OF MORAX-AXENFELD.

Dr. Brown Pusey reported ten cases of infection of the eye with the diplobacillus of Morax-Axenfeld observed in Chicago during the last six months. Five of these cases were cases of chronic conjunctivitis, in one of which there had been marginal ulcers of the cornea; three were cases of acute conjunctivitis; one was a severe ulcer of the cornea, and in one case there were no objective or subjective symptoms.

These being the first cases recorded in Chicago, of infection by this organism, Dr. Pusey reported the clinical histories of his cases in some detail, and spoke of the characteristics of the organism and exhibited smears and cultures of it. From his experience he concludes that the Morax-Axenfeld bacillus is more widespread in this region than has been supposed.

DISCUSSION.

Dr. N. M. Harris called attention to the tendency to specialization in bacteriology, and quoted the dictum of Eyre that the diagnosis of inflammations of the eye should not rest so much on the clinical findings as on the bacteriology of the exudates. For not only is the number of organisms causing specific inflammations of the eye relatively small and their cultural reactions easily determined, but their morphological differences well marked as well. The organism under discussion is best called, by Eyre, the *Bacillus lacunatus*, because of its peculiar habit of forming holes or lacunæ on coagulated blood serum. It is very similar to, and perhaps the same organism, as that described by Pettit as growing on ordinary media and liquefying gelatin; indeed Axenfeld and Eyre both have found that subcultures of the *B. lacunatus* may acquire these characteristics.

Dr. F. A. Phillips exhibited a probable case of this form of conjunctivitis. It has not yet responded to the cultural tests, but the microscope has demonstrated the presence of the bacillus on two occasions. When first seen there was a very marked erosion of the lids at the inner and outer canthus. The secretion was scanty and light in color. The patient has had the condition for over a year. Simple antiseptic treatment has been instituted until cultures of the organism shall have confirmed the clinical diagnosis.

In the case of an infant seen some months ago a serious ophthalmia was suspected, but the microscope showed the presence of the diplobacillus, although no cultures were made. Active treatment was instituted with good results.

Dr. H. V. Würdemann:—Dr. Pusey's paper points out the necessity of making office examinations of the secretions from the conjunctiva. The diagnosis is made easily and the treatment is outlined positively in each instance, whether the case is one of diplobacillary conjunctivitis or gonococcus or streptococcus conjunctivitis. If there is any doubt, the services of a bacteriologist can be secured or a blood serum tube may be inoculated and sent to the bacteriologist for diagnosis. The silver salts are specific for the gonococcus and the zinc compounds are specific for the Morax-Axenfeld diplobacillus.

He cited the case of a boy who had been treated for an acute purulent conjunctivitis for over a week; he was convinced that gonorrheal infection was entirely out of the question, and a microscopical examination of the secretion at once demonstrated the presence of the diplobacillus. No other germs were found. Sulphate of zinc cured the case in three days. A few days later the father came with the same condition. The smears showed the diplobacillus, and sulphate of zinc produced prompt results.

Dr. W. E. Gamble cited the case of a boy seen about a month ago who had an acute conjunctivitis in the right eye, without much discharge. A smear was made and it showed the diplobacillus. Proper treatment was instituted, after having used argyrol without effect, and the eye got well.

Another case of ulcer of the cornea was treated for months, and yet the eye is no better. Argyrol and boric acid solution proved ineffective. An optical iridectomy was done, but the operation was not very successful. Smears were not made in this case, except early in the disease.

He has recently seen four cases of acute conjunctivitis in which the ocular conjunctival injection was quite marked. The microscope showed the diplobacillus, so that the picture need not necessarily be of the chronic type. The disease may be acute in character. However, that may be due to a mixed infection.

Dr. E. V. L. Brown said that it is possible to make a diagnosis of this condition in many cases from the clinical features alone. A mixed infection may, however, change the picture of a chronic disease into that of an acute affection. In the case cited by Dr. Phillips two other organisms were found, a staphylococcus and another diplococcus.

He has observed in the laboratory that zinc sulphate is not a specific for this form of conjunctivitis, as has been claimed. Some patients have been under treatment for two or three months, although it may be possible that they are not observing the instructions given by the physician as closely as is the case with a private patient. But in all these cases the diplobacillus is still present in the secretions.

Dr. Pusey stated that in all his cases after using the zinc the results were apparent immediately; but all his patients were private patients, except two who were in a hospital, and even over these he had absolute control, and as Dr. Brown stated, he thought that this might make some difference in the results obtained from treatment.

As to the nomenclature of the bacillus, he protested at the bacteriologist taking away the little credit the ophthalmologist has earned in this connection, because Morax and Axenfeld are ophthalmologists, and, therefore, it is well to have their names connected with the organism.

Meeting, May 8, 1906.

DIONIN IN OLD CORNEAL OPACITIES.—DRS. C. W. HAWLEY AND M. Z. ALBRO.

The writers reported four cases of old corneal opacities and infiltrates, markedly cleared by long continued use of 2 to 10 per cent. solution of dionin.

Cases 1 and 2.—Mother and son, respectively, had 5 mm. and larger, prominent, white opacities; only one small opaque point at the present time can be seen in the boy's cornea, and one must use a lens to discover the infiltrate whatever in the mother's cornea.

Case 3 had a very dense 2x3 mm. scar, evidently due to extensive ulceration, and a narrow band of infiltration in addition to general opacity almost entirely hiding the iris; under treatment a 3 mm. clear marginal zone has appeared through which the iris is plainly visible and the denser scars much smaller and thinner.

Case 4 is a leucoma existing from childhood, and is gradually disappearing under treatment.

DISCUSSION.

Dr. Oscar Dodd compared the results to be obtained from the use of dionin in one eye and jequiritol in the other. In his experience, the latter gives the better results in inflammatory conditions and the former in the non-inflamed opacities, although dionin seems to work the better in mild inflammatory conditions. In opacities due to corneal ulceration, he has obtained very little benefit from jequiritol.

Dr. W. E. Coleman has found that the eye becomes tolerant to dionin after 5 or more days of use; galvanization before its use increases its effectiveness. He used this treatment in an acute case of iridocyclitis, with rapid clearing of the cornea.

In the case of a child six months old, whose cornea was injured during a forceps delivery, he used dionin faithfully for six months without any effect whatever.

Dr. Hawley had obtained no favorable results in these cases, either from the use of yellow oxid of mercury and massage, or electricity; his results had been obtained exclusively with dionin.

Dr. J. Elliott Colburn reported the case of a woman, aged 32, who had a large central opacity following ulcer of the cornea that occurred during an attack of smallpox when the patient was six years of age. It was quite dense, but the curvature of the cornea was symmetrical. He used dionin for about two months without any perceptible clearing of the cornea. Having read in some journal that the cornea would clear more rapidly when the opacity is somewhat recent, it occurred to him that he might create a more recent condition, so he applied electrolysis, searing the entire surface of the corneal scar. After the condition had passed the acute stage, he used dionin with the result that the patient acquired about 20/40 vision within four or five months.

Dr. F. E. Brawley:—"Affection of the Frontal Sinus and Anterior Ethmoidal Cells Manifesting Purely Ocular Symptoms." (Membership Thesis.) The writer gave a detailed report of 8 cases in point, along with a résumé of the literature; headaches beginning about the eye, and accompanied by inability to use the eye during the attack, are practically always of nasal origin, involving the frontal and ethmoidal sinuses, and usually non-suppurative in type.

2. The condition is best explained by supposing that turgescence of the middle turbinal of the affected side causes an imprisonment of the air in the frontal and anterior ethmoidal cells by blocking their exits into the nose. Absorption of the contained oxygen by the lining membrane of the cells brings about a vacuum, congestion and swelling of the membrane follow, producing irritation of the contained nerve endings, branches of the ophthalmic division of the fifth nerve with resulting reflex ocular disturbance. An acute congestion occurs also in the orbital tissues as a result of the blockings of the branches of the ophthalmic artery which supply the lining membrane of these cells.

3. The application of adrenalin to the affected middle turbinal is a specific means of diagnosis of this condition by reëstablishing air communication between nose and sinuses with resulting relief of subjective symptoms.

4. A specific treatment is the resection of the anterior third of the affected middle turbinal with consequent exposure of the exits from these sinuses and permanent relief from the subjective symptoms. (See *Ophth. Record* for complete article.)

DISCUSSION.

Dr. N. M. Black, Milwaukee, reported two cases similar to those cited by Dr. Brawley. (See *N. Y. Med. Jour.*, June 2, 1906.)

Dr. Brown Pusey directed attention to the fact that relief might have been afforded these patients merely by the bleeding from the turbinates, as this has been known to occur quite frequently.

Dr. F. A. Phillips stated that it must not be forgotten that there exists a relationship between lowered vision and hysteria, and that it is necessary to take the fields and make other observations in these cases before hysteria is excluded. The relief that is afforded from the simple application of adrenalin is easily accounted for in the class of cases known as hysterical amblyopias and asthenopias. He thought that Dr. Black's cases went through the typical cycle of hysterical manifestations. There was no original disturbance to account for the symptoms present, simply a functional disturbance, hysterical in character.

Dr. Black replied that on examination of the fundi of his patients nothing abnormal was found, except when the electric ophthalmoscope was used, when he clearly saw a peculiar punctate condition about the macular region, fine yellow spots, fifty or sixty of them, extending over an area about the size of the disc. These spots had disappeared entirely after the vision had become normal.

Dr. Brawley agreed with Dr. Phillips as to the necessity of making more careful and thorough examinations of the fundi and eye grounds of this class of cases. He was not ready to believe that these cases were hysterical amblyopias. In his own case the symptoms were always the same and the results obtained from treatment were always identical. His symptoms were exactly the same as those experienced by the patients whose histories he reported.

A FRAGMENT OF STEEL IN THE EYE.

Dr. Wm. H. Wilder reported the case of a young man who was working with a hammer and cold chisel when a piece of steel flew into the left eye. There seemed to be some irritation of the lid and a small wound, about one and a half millimeters in length, was seen just within the limbus at the nasal side. Examination of the fundus revealed a scar or track in the interior temporal quadrant of the choroid at the end of which was a black object, apparently a little mass of pigment, which at that time was supposed to be a foreign body. A little blood vessel ran across this track. With the large magnet the piece of steel was brought up to the wound, yet it could only be extracted after the wound had been enlarged slightly with Knapp's discission knife; then even the metal could not be withdrawn with the Hirschberg magnet, because, when the tip of the magnet was withdrawn, the foreign body stripped off. With the large magnet the fragment was, however, easily withdrawn. Recovery has been uneventful and without reaction; the media are perfectly clear and the object in the choroid which was mistaken for a foreign body, can still be seen; vision has returned to normal.

ANOTHER SUCCESSFUL MAGNET OPERATION WITH AN UNUSUAL TERMINATION.

Dr. H. B. Young, Burlington, Iowa:—The patient presented a 6 mm. penetrating corneal wound caused by a piece of steel striking the eye an hour before; a tag of iris incarceration was removed and a piece of steel 6 by 7 by 1 mm. in size removed, through an enlarged incision, by the Haab magnet after failure with the hand magnet. The wound healed promptly, although some iritis supervened; the eye was quiet a month later, with qualitative vision and doubtful light perception; the tension was distinctly lowered, though there was no ciliary tenderness. A week later the patient suddenly became wildly excited, then maniacal, incoherent, and finally oblivious to his surroundings, and died on the sixth day, apparently from exhaustion; his temperature and pulse were normal throughout. At autopsy the cavities of the skull, thorax and abdomen were opened and the contents found macroscopically normal. The eye, with a section of the nerve, was removed and given to Dr. Brown Pusey for further study.

A BACTERIOLOGICAL STUDY OF FIFTY CASES OF CONJUNCTIVITIS FOUND IN CHICAGO DURING THE PAST YEAR.

Dr. Brown Pusey reported the results of some work done in Chicago during the last year on the bacteriology of the normal and diseased conjunctiva. He finds the Gram positive bacillus of the group pseudo-diphtheria or xerosis almost constantly

present in the conjunctival sac. He has not seen a case where this organism could have been considered the pathogenic agent. Staphylococci are very frequently found in the conjunctival sac; in Chicago one seldom makes a smear or culture from the conjunctival sac without finding staphylococci. He found staphylococci where no inflammatory symptoms existed and in cases of conjunctivitis where there was little question that this organism was the pathogenic agent. The organism most frequently found as the cause of acute conjunctivitis in Chicago is the pneumococcus. Dr. Pusey has found 12 such cases in the year. These cases were sporadic; so far as he knows there has been no epidemic from this organism in Chicago during this time. He had four cases of acute conjunctivitis caused by the influenza bacillus; three of these cases were in one family and recovered in three or four days without treatment. He has had in this time three cases of gonorrheal infection of the eye. One of these cases was in an elderly woman and the mild character of the inflammatory reaction was remarkable. He found infection of the conjunctival sac by the diplobacillus of Morax-Axenfeld in eleven cases. Seven of these were cases of chronic conjunctivitis, three were cases of acute conjunctivitis, and in one there were no symptoms. He has been actively on the lookout for the Koch-Weeks bacillus, but has not seen it in Chicago. In concluding he called attention to the facts that the pneumococcus appears to be the most frequent cause of acute conjunctivitis in Chicago and that the diplobacillus of Morax-Axenfeld is the most frequent known infectious agent giving rise to chronic conjunctivitis. He emphasized one point brought out prominently by his series of cases, namely, that one must be most careful about venturing an opinion regarding the nature of the infectious agent from the clinical findings; in his cases he had a severe case of acute conjunctivitis caused by the Morax-Axenfeld diplobacillus and a case with very slight reaction caused by the gonococcus.

E. V. L. BROWN, *Secretary.*

GREENE COUNTY.

The annual meeting of the Greene County Medical Society was held in Mercantile Hall, Whitehall, Ill., Friday, Sept. 14, 1906. Called to order by President Chapman. Minutes of the previous meeting were read and approved. In the absence of Dr. F. H. Russell, member of the board of censors, Dr. H. W. Hand was appointed to act with Drs. Howard, Burns and Gooch. The application for membership of Dr. Royal of Wrightsville having been presented at a previous meeting, was recommended by the board of censors, and, on motion of Dr. Hand, the secretary was instructed to cast the ballot of the society for the applicant. The election of officers for the ensuing year resulted as follows: President, Dr. H. W. Chapman; first vice-president, Dr. F. H. Russell; second vice-president, Dr. Jas. Squires; secretary and treasurer, Dr. H. A. Chapin.

The board of censors reported on the application of Dr. E. K. Shirley, Whitehall, Ill.; Dr. L. O. Hamilton, Barrow, Ill.; and Dr. H. W. Smith, Roodhouse, Ill., for action at the next meeting. Whitehall, Ill., was recommended as the next place of meeting, Dec. 14, 1906. The essayists appointed for the next meeting are: Dr. J. W. Adams, Carrollton; Dr. F. H. Russell, Eldred; Dr. H. Converse, Greenfield; Dr. E. H. Higbee, Roodhouse; Dr. H. A. Chapin, Whitehall. On motion the report of the board of censors was unanimously adopted.

Dr. Percy, of Galesburg, President of the Illinois State Medical Society, then read a most interesting and instructive paper on The Borderland of Insanity, which was ably discussed by the members and visitors. After the discussion Dr. Percy explained in detail the plans of the committee on medical defense, setting forth its advantages.

The question of life insurance examinations was discussed and the following resolution adopted:

Resolved, That we, the undersigned members of the Greene County Medical Society, and all other reputable physicians of said county, do hereby agree, by subscribing our names to this resolution, that we will not make an examination for any old line life insurance company for less than five dollars ($5.00), nor

for any mutual or fraternal insurance company for less than two dollars ($2.00), and that any physician who would violate this resolution would be deemed unworthy to be met in counsel.

The secretary was instructed to present the resolution to each physician in the county for his approval and signature.

On motion of Dr. Squires the thanks of the society were extended to Dr. Percy for his attendance and excellent paper.

After remarks regarding the future work, the society adjourned. Those present were: J. F. Percy, Galesburg; Howard Burns, E. S. Gooch, J. W. Adams, James Squires, Carrollton; E. H. Higbee, H. W. Smith, Roodhouse; A. Royal, Wrightsville; L. O. Hamilton, Barrow; H. W. Chapman, A. W. Foreman, J. W. Redwine, G. W. Burns, E. K. Shirley, H. A. Chapin, W. C. Day, H. W. Hand, Whitehall.

H. A. CHAPIN, *Secretary.*

LAWRENCE COUNTY.

The Lawrence County Medical Society met in regular session Sept. 3, 1906, with the following members present: Drs. Z. D. French, J. B. Bryant, A. G. Mountz, H. V. Lewis, J. E. Emmons, F. F. Petty, H. K. Murphy, B. F. Hockman and Chas. P. Gore. Papers were read by H. K. Murphy, of Sumner, and F. F. Petty, Chauncey. Both papers were of value to the society, and all members present took an active part in the discussion. One of the important things the society did in the matter of business was to vote unanimously to accede to all the requirements of the state society in the matter of dues This meeting was one of the best the society has ever had.

B. T. WORKMAN, *President.*
CHAS. P. GROVE, *Secretary.*

VERMILION COUNTY.

The Vermilion County Medical Society met September 10, in the City Hall at Danville. The paper of the evening was on Functional Disorders of the Stomach, by Robert McCaughey, of Hoopeston. He briefly reviewed the anatomy and physiology of the stomach, followed by a demonstration of stomach contents. He showed how absurd it is to attempt diagnosis of a stomach disorder without a stomach analysis. One might as well attempt to diagnose a kidney lesion without a urinalysis. The discussion was opened by A. M. Miller of Danville. Dr. J. H. McIntosh proposed that the physicians unite on a plan of collecting accounts and keeping track of "dead beats." The chair appointed a committee to present this to the physicians and have some more definite information by the next meeting. Adjourned.

E. E. CLARK, *Secretary.*

OBESITY.

W. C. ABBOTT, M.D.
CHICAGO.

At the last meeting of the Illinois State Society, Dr. Croftan contributed a suggestive paper on obesity (ILLINOIS MEDICAL JOURNAL, Sept, 1906). Speaking of the treatment, he mentioned diet, hydropathy, exercise, sanatoria, and limited the use of drugs strictly to thyroid extract. To this he also made the grave objection that its use sometimes developed glycosuria, a thing particularly to be dreaded in the obese, since they are already predisposed to diabetes. That thyroid extract occasionally causes disagreeable, and even dangerous symptoms, and is uncertain and transient in its effects, may be set down to lack of care and skill in its administration. But—is there nothing in obesity but overeating, as compared to the need of food?

In the discussion Dr. Elliott spoke of the general excess of vascular tension in the obese; and, while he affirmed that obesity was only an error of diet and nothing more, in the next breath he contradicted this by saying that, in many cases, there

was a morbid disposition to lay on fat, the weight increasing even when the caloric value of the food is low, its quantity or bulk small.

Dr. Marcy confirmed the statement that many obese persons eat little, but said the one important thing is graduated exercise. Dr. Webster confirmed the occurrence of obesity in small eaters and looked on them as especially liable to diabetes. Dr. Mettler classed obesity as a tropho-neurotic malady. Dr. Fairbrother insisted on diet, but added cathartics.

We can not look on this malady as so simple a matter. There is a reason why one man will gain flesh, while eating and drinking less than another man who remains thin. Granting the necessity of diet and exercise in the reduction of fat, there are other elements in the disease, and, consequently, other indications to be met, and, if we comprehend these, we may be able to meet them with appropriate remedies. That we can not do this signifies a lapsus in our knowledge of pathology and therapeutics.

But we may reason back to a certain extent. If we find a definite pathologic state present, and ascertain empirically that a certain medicament will remove the symptoms and restore healthy equilibrium, we may assume that it does so by removing the pathologic conditions that produced the evident symptoms. We do not know how mercury cures syphilis, and yet we know that it does cure it. Similarly, we know that phytolaccin will, in many cases, materially reduce the weight of the obese by causing the disappearance of the fat. The difficulty in general recognition of this fact is that the effect does not at once follow, but is usually not manifested until the drug has been taken for more than three months. When the absorption of fat sets in, it is unmistakable.

The best results follow when the diet has been arranged and the exercise has been exactly fitted to the case, phytolaccin, in doses well borne, has been given, and, to this, moderate doses of thyroid extract are added. Leave out either of the four remedies and the results are not as good, at least, the reduction is slower. This is a good method for the first month, when a reduction of material dimensions may be secured, which will reconcile the patient to persistence in treatment. After this, it is usually best to dispense with the thyroid extract and continue with the other remedies, being satisfied with a moderate reduction of not more than five pounds a month. This is safe, and may be accomplished without more interference with the patient's diet and habits than he will submit to without feeling his restrictions unbearable.

NEWS OF THE STATE.

Dr. Lister has located at Cabery.

Dr. Bowden Bird has located at Salem.

Dr. Albert Purcell has located at Streator.

Dr. Thomas Pickle has located at Mill Creek.

Dr. C. E. Gwinn, of Oakland, has located in Sadorus.

Dr. C. C. Kost has removed from Dixon to Freeport.

Dr. W. O. Beam, of Moline, has moved to Davenport, Iowa.

Over thirty cases of diphtheria are reported from Spring Valley.

Dr. E. O. Hutchins has removed from Keokuk, Iowa, to Warsaw.

Dr. Wilbur J. Hawkins, Aurora, is ill at his home with appendicitis.

Dr. J. W. D. Mayes, of Illiopolis, has moved into a beautiful new home.

Dr. Albert S. Wall, Champaign, has returned after four months in Europe.

Dr. and Mrs. Bernard Fantus, of Chicago, have returned from Europe.

Dr. J. E. Huston has taken an office in the Odd Fellows' Building, Danville.

Dr. J. M. Coleman, of Delavan, has been appointed physician for Pekin Township.

Dr. J. S. Cunningham, of DesPlaines, has removed to 6527 Normal avenue, Chicago.

Dr. William C. Hill, Murphysboro, is reported to be critically ill with typhoid fever.

Dr. B. B. Griffith, of Springfield, spent the month of August at Colorado Springs.

Dr. M. W. Snell, of Litchfield, has recently been made the cashier of the bank in that city.

Dr. Lewis H. Skaggs, Ellsworth, was overcome by the heat, August 23, but is improving.

Drs. Jeremiah H. Stealey and Carl F. Snyder will establish a modern sanitarium in Freeport.

Dr. and Mrs. Lyman Ware, Chicago, have returned from a four months' trip in Europe.

Dr. and Mrs. Julien E. Hoquembourg have returned after a month's trip to the Pacific coast.

Dr. George A. Wash, of Palmyra, is in Ohio, with a view of becoming a resident of that state later on.

Dr. Amos S. Bickel, North Chillicothe, is seriously ill with anemia at St. Francis' Hospital, Peoria.

It is reported that the schools at Bushnell have been closed on account of an epidemic of diphtheria.

Dr. Clinton G. Lumley, who was operated on for appendicitis August 28, is making good progress toward recovery.

During August, the Visiting Nurses' Association, Chicago, cared for 1,227 patients and the nurses made 5,738 visits.

Dr. E. H. Leib has removed to Springfield and has taken offices with his brother, Dr. J. R. Leib, in the Myers Building.

Dr. George E. Baxter, Chicago, was held up and robbed of a gold watch and a small amount of money, September 12.

Dr. E. R. Herrmann has purchased the practice of Dr. J. S. Cunningham, recently of DesPlaines, and will locate there.

Dr. Lawrence A. Mendonsa, a graduate of the St. Louis College of Physicians and Surgeons, has located in Springfield.

Dr. Frederick Tice, of Chicago, has returned from abroad and resumed practice in the Columbus Memorial Building.

The Swedish-American Hospital Association, Green and Sixtieth streets, Chicago, has borrowed $35,000.00 for improvements.

Dr. Fred D. Hollenbeck has removed to 4411 North Ashland boulevard, Chicago. His office remains at 394 East Chicago avenue.

Dr. C. J. Eads, of Oquawka, has sold his practice to Dr. C. E. Kaufman and has discontinued the practice of medicine for the time being.

Dr. Bellwood, of Abingdon, narrowly escaped death from strychnin poison recently. A large dose of the medicine was taken by mistake.

Dr. Albert H. Dollear, Jacksonville, has been appointed assistant physician of the Illinois Western Hospital for the Insane, Watertown.

Dr. Henry W. Cheney, 369 East Sixty-third street, Chicago, has returned to the city, after an eight months' absence in Berlin and Vienna.

Dr. Maximilian Herzog, who recently returned from the Philippine Islands, has been elected pathologist to the Michael Reese Hospital, Chicago.

Dr. Christian R. Hartung was examined in the insane court, September 6, and committed to the Cook County Hospital for the Insane, Dunning.

Dr. Carl E. Black, of Jacksonville, chairman of the Publication Committee of THE JOURNAL, has returned from a summer vacation trip to Germany.

The Board of Health of East St. Louis has instructed the health inspector to see that conductors of street cars enforce the laws regarding expectoration.

An epidemic of diphtheria at Bardolph, near Bushnell, has grown to such proportions that the authorities have ordered all schools and churches closed.

The Annual Physicians' Golf Tournament was held at Glenview, September 18 and 19. A large number of Chicago physicians participated in the tournament.

Dr. Roskoten, 200 South Jefferson avenue, Peoria, has sold his property in that city and will remove to the State of Washington, where he will engage in practice.

Dr. R. A. McClellan and wife, of Yorkville, started for New York, September 22, for a three months' stay. They will spend most of the time in London and Berlin.

The Illinois State Board of Health has begun a campaign against the fake doctors of the State. During the month of August, fifty-six prosecutions were instituted.

The King of Portugal has conferred on Dr. Alexander Hugh Ferguson, of Chicago, a commandership in the Order of Christ of Portugal in recognition of his original work in surgery.

Chief of Police Bargren, of Rockford, is making an effort to prevent the distribution of pamphlets, advertising patent medicines, which have been given out by the free distributors in his city.

Dr. Edwin Kuh, 4330 Drexel boulevard, Chicago, has begun suit against Benjamin R. Cahn for $3,000.00, said to be due him for professional services rendered during the past six years.

The twelfth annual commencement exercises of the Illinois Medical College were held August 23, when degrees were conferred on a class of fifty by Dr. Heman H. Brown, president of the college.

Dr. Alfred C. Girard, United States Army, retired, has been obliged to give up his plan of practicing in Chicago on account of the serious illness of his wife, and has returned to the Pacific coast.

Dr. P. J. H. Farrell, of Chicago, has been nominated for the Board of Review of Cook County on the Independence League ticket. Dr. B. S. Turner has been nominated for assessor on the same ticket.

It has been announced that Dr. D. K. Pearsons has donated an additional $5,000 toward the fund for the new Presbyterian Hospital, which brings the total fund to within $10,000 of the $400,000 required.

St. Vincent's Hospital, Taylorville, was dedicated, August 22, with appropriate ceremonies. The hospital was erected at a cost of about $25,000 and will be in charge of the Sisters of the Most Precious Blood.

Dr. F. G. Hall, Burlington Relief Medical Examiner at Galesburg, has resigned his position with the company to go into private practice. He will be succeeded by Dr. A. O. Early, of Grand Crossing, Wisconsin.

Dr. Wm. A. Garner, of Clayton, has been sued for $10,000.00 damages by the administrators of the estate of Roy Johnson, who died in August, 1905. It is claimed that the young man's death was due to infection.

Comptroller McGann has received an offer from a woman philanthropist to erect a school for the blind on the Gage farm. The donor proposes to make the school non-sectarian and expects to put up a building to cost about $50,000.

The Lutheran-Norwegian Deaconess Hospital has appointed the following staff: Surgical, S. Dahl, A. Holmboe, B. E. Henderson, J. V. Fowler, J. H. Burwash; medical, M. T. Quales, E. J. Hook, J. R. Ballinger, A. B. Oyen and Carl Rolleason.

Dr. Podstata, who was recently appointed superintendent of the Elgin Asylum, has announced that he will hereafter discharge any em-

ployés who indulge in gossip. It is reported that Samuel Case, the druggist, has been discharged for this reason.

A joint meeting of the various state boards was held at Kankakee, on July 31, at which it was decided to make a number of changes in the conduct of the Eastern Hospital for the Insane. A training school for nurses and attendants will be established, and improvements in the system of nursing are contemplated.

The Illinois State Board of Health has issued a bulletin for August, containing a large amount of interesting matter. Considerable space is devoted to the matter of prosecution of the violators of the Medical Practice Act and to itinerant vendors. Many other matters of interest to the profession in the State are also considered.

Dr. Vaclav H. Podstata, superintendent of the Illinois Northern Hospital for the Insane, Elgin, announces the opening of a training school for attendants and nurses on October 1. The school will have a three years' course, and the attendants will be given more time off duty and the hours in various quarters will be shortened.

Dr. Frederick W. Whitney, said to be connected with the Louis Chemical Company, was arrested by United States Deputy Marshall Wainwright, at Chicago, on September 22, on a warrant charging misuse of the mails. Postoffice inspectors state that a certain formula has been advertised through the mails in violation of the law.

The Coroner of Cook County reports, for August, a larger death rate than any month since the Iroquois Theater fire. The table of deaths from other than natural causes during the month was as follows: Suicide, 39; drowning, 29; heat prostration, 27; railroad accidents, 26; homicide, 14; street car accidents, 13; lightning, 1; scenic railway accident, 1.

As the result of a conference held between the Secretary and Assistant Secretary of State Board of Health and the directors of the State Board of Water Survey, Urbana will probably have, in a short time, a modern septic tank for the purpose of demonstrating to the officials of the various municipalities the advantages of this method of sewage disposal.

Anton P. Freund, druggist at St. Luke's Hospital, has been discharged from that institution as a result of his mistake in furnishing atropin instead of a proprietary preparation of a somewhat similar name in filling a prescription for a patient in the hospital, who died soon after. The coroner's jury recommended that Freund's license as pharmacist be revoked.

A new site has been provided for the Chicago Lying-in Hospital and Dispensary, now located at 294 Ashland boulevard, at the northwest corner of Vincennes avenue and Fifty-first street. The cost was $52,500, and the new hospital will be erected at a cost of between $150,000 and $200,000. The new hospital, with 60 beds, will be able to care for 1,200 patients a year.

Some of the residents of Niles Center have been victimized, it is reported, by "Doctor" Robt. H. L. Saunders, who located in that village a short time ago. It is stated that Dr. Saunders announced that he was heir to a large fortune left him by his uncle, which would come into his

possession as soon as he was married. After being the social lion of the village for several weeks, he is said to have left the town.

Dr. B. S. Dennison, formerly of Salida County, Colorado, was arrested in Chicago, September 22, charged with arson and conspiracy, and gave bond before Judge Prindiville. It is alleged that he was implicated in setting fire to a jewelry store in order to defraud the insurance companies. Dr. Dennison asserts that the charges are false and are part of a conspiracy to obtain control of a copper mine in which he is interested.

The members of the Chicago Tuberculosis Institute have perfected plans for the erection, at Dunning, of portable houses for patients suffering from tuberculosis, who will be given treatment at the new camp, which is now being established. The houses consist of dormitories, 26 by 44 feet, and include two porches, a kitchen and a dining room. Three of the cottages are now ready and the camp, with accommodations for 20 or 30 patients, will probably be opened this week.

Chas. Passow & Sons, manufacturers of billiard tables and saloon fixtures, have complained to the Chicago Health Department that the Jenner Medical College, 196 Washington street, has been burning cadavers in the furnace in the basement of their building, and that the health of the occupants of the building was thereby being endangered. This statement is denied by the registrar of the school, who says that the charge is the result of a quarrel between one of the Passow brothers and the college janitor.

Typhoid fever is reported to be prevalent in Alton, and the necessity of boiling all drinking water is urged. Joliet reports four cases of scarlet fever. Diphtheria has been prevalent in Brereton, and the situation is being investigated by an official of the State Board of Health, who recommended that every house in which there had been a case of diphtheria should be properly disinfected and fumigated, and that all clothes and wearing apparel should be disinfected. He ordered the two families at present under quarantine to be released and the houses fumigated.

The State Board of Charities, in its quarterly report, just issued, states that since the organization of the board, last April, the commissioners have visited every hospital for the insane in the state, 52 county almshouses, 52 jails, 19 orphanage and child saving institutions; it has investigated the charges against the Soldiers' Orphans' Home, Normal; the Soldiers' Widows' Home, Wilmington, and the Asylum for Feeble-minded Children, Lincoln, and has inspected the schools for the deaf and blind at Jacksonville, and the Soldiers' and Sailors' Home, Quincy.

The Health Department of Chicago has discovered 6 cases of typhoid fever, one of which was fatal, between Morton street and Tuohy avenue, Rogers Park. In this location, the residents have no city water service, and are obliged to depend on well water or purchased water for drinking or household purposes. About one hundred cases have been reported to the Board of Health in various parts of the city. The officials declare that the milk and ice supply is responsible for the outbreak of this disease. Ice companies have run short and have been compelled to import ice

from the country that was cut from ponds filled with stagnant and impure water.

At the annual convention of the Illinois State Coroners' Association, held in Chicago, September 11, 48 counties were represented. Peter M. Hoffman, coroner of Cook County, was elected president of the association; C. Z. Noel, Joliet, and C. N. Streeper, Alton, were elected vice-presidents; R. L. Baker, Peoria, was elected secretary, and William Woodruff, Springfield, treasurer. One of the most important matters brought up was the necessity of passing a law compelling physicians to report cases of a certain class to the coroner. There is nothing at present in the statutes to prevent a physician of Illinois from signing a death certificate, although he may not have attended the deceased in the capacity of physician for a number of years.

The State Board of Charities, in its quarterly bulletin, states that, as a result of its inquiries, the board feels that there is pressing need for a correlation of the energies of the state hospitals for the insane. No two institutions are alike, but in the majority of items of administration standardization is necessary to elevate the institution to a high plane of efficiency and economy. The hospitals for the insane were found to be top heavy on the custodial side. The superintendents are burdened with business details which prevent them giving sufficient attention to the medical administration. For this reason, the hospitals are more like detention boarding houses than like places for the modern treatment of hopeful cases and the industrial re-education of chronic types.

The American Surgical Trade Association requests the insertion of the following notice regarding a uniform scale for all catheters, bougies and sounds: At a meeting of the American Surgical Trade Association, held in Philadelphia, June, 1906, it was resolved that after January 1, 1907, the trade adopt the French scale for all catheters, bougies and sounds. A committee was appointed for the purpose of getting up a proper and accurate French scale card, and the same will be mailed to you. Every physician will see the importance of this step, as you are all acquainted with the annoyance of having catheters, bougies and sounds and other instruments marked in American, English or French numbers. You are requested from above date to use only the French scale in ordering such goods and when no scale is specified orders will be filled by the French scale.

The Illinois Coroners' Association will meet in Springfield in November to draft a bill for presentation to the next legislature. The legislative committee of the State Association is composed of the following members: Peter Hoffman, coroner for Cook County; Dr. Leslie Baker, coroner of Peoria County; Wm. Woodruff, coroner of Sangamon County, and Coroner Streeper, of Madison County. The bill will include the following points: 1. Witnesses before a coroner's inquest are to be required to answer questions put by the coroner. 2. Autopsies to be held and paid for on the order of the coroner. 3. Coroners to be allowed a per diem fee when inquests extend over more than one day. 4. Physicians to be compelled to report cases to the coroner, under penalty for failure to do so.

5. A penalty provided for moving or disturbing a dead body without authority from the coroner. 6. Undertakers are not to inject embalming fluids into a body without the coroner's permission. 7. Undertakers are not to use any fluid containing arsenic or strychnin under any circumstances. 8. Coroners are to be allowed the same fee as sheriffs for serving subpœnas.

The Military Tract Medical Association will hold its next annual meeting at Macomb, Oct. 18, 1906. The officers are as follows: President, R. C. Matheny, Galesburg; first vice-president, W. E. Shellenger, Canton; second vice-president, J. P. Roark, Bushnell; secretary-treasurer, F. E. Wallace, 322 Warren avenue, Chicago; board of censors, J. B. Bacon, Macomb; S. M. Miller, Peoria; M. T. Rogers, Cuba.

Committee on Necrology, W. E. Griggsby, Blandinsville.

Program:

1. President's Introductory Address—R. C. Matheny, Galesburg.
2. The Regulation of the Medical Profession—Hon. L. Y. Sherman, Lieutenant Governor of Illinois, Macomb.
3. Non-operative Treatment of Pelvic Infection in Women—T. J. Watkins, Chicago.
4. Cancer of the Skin, Illustrated with Stereopticon—Wm. A. Pusey, Chicago.
5. Retention of Urine: Its Causes and Treatment—J. A. Patton, Chicago.
6. Modern Neurological Diagnosis—L. H. Mettler, Chicago.
7. Problems in Infant Feeding—T. G. Allen, Chicago.
8. The Ultimate Result in Hip Disease—J. L. Porter, Chicago. Illustrated by *x*-ray pictures and photographs.
9. Euphysema of the Maxillary Antrum—A. M. Corwin, Chicago.
10. Subject Not Announced—A. McDermid, Chicago.
11. Subject Not Announced—J. A. Egan, Springfield.
12. Subject Not Announced—L. T. Thurman, Macomb.
13. Pleasant Medication—Bernard Fantus, Chicago.
14. The Limitations and Requirements of the Modern Treatment of Tuberculosis—J. W. Pettit, Ottawa.
15. The Dumb-bell Intestinal Anastomosis—J. B. Bacon, Macomb.
16. Vibration—M. S. Marcy, Peoria.
17. Postoperative Adhesions—L. S. Stremmel, Macomb.

MARRIAGES.

Dr. H. B. Milhon and Miss Rosella Clawson, both of Owaneco, Oct. 2, 1906.

Frank A. Hill, M.D., to Miss Fidele A. Madison, both of Chicago, August 15.

Charles Burr Caldwell, M.D., to Miss Lydia Stevenson, of Monticello, Ill., August 30.

B. F. Zobrist, M.D., Jewett, Ill., to Miss Lillie A. Apple, of Keysport, Ill., September 4.

Dr. Irving W. Metz, of Springfield, and Miss Edith Thorp, of Havana, Sept. 26, 1906.

John A. Cavanaugh, M.D., Chicago, to Miss Irene Cummings, of Rock Valley, Iowa, September 12.

R. B. Hoag, M.D., Lincoln, Ill., to Miss Hephzibah E. Green, at South Pasadena, Cal., September 6.

Richard H. Street, M.D., Chicago, to Miss Lillian Woodward Gunckle, of New York City, August 27.

Henry Bascom Thoman, M.D., Chicago, to Miss Louise Downing Wendell, of Louisville, Ky., September 5.

Anson Le Roy Nickerson, M.D., Momence, Ill., to Miss Laura May Chesney, of Bucyrus, Ohio, September 12.

DEATHS.

Joseph Wolfgarten, M.D., National Medical University of Chicago, 1895, died at his home in Chicago, September 10.

Edward L. Birch, M.D., Miami Medical College, Cincinnati, 1879, died at his home in Robinson, Ill., August 7, from ptomaine poisoning, after an illness of two months.

George W. Fellows, M.D., Vermont Medical College, Woodstock, 1851, for many years a practitioner of Kewanee, Ill., died at his home in Creston, Iowa, September 6, from Bright's disease, after a long illness, aged 78.

Maurice James Chase, M.D., Vermont Medical College, Woodstock, 1850, for half a century a prominent practitioner of Galesburg, Ill., died at the home of his daughter in that city, September 6, after a brief illness, due to the effects of a fall, aged 80.

Christopher Anton Jaeger, M.D., Western College of Homeopathic Medicine, Cleveland, 1864, a veteran of the Civil War, said to be the oldest practitioner of Elgin, Ill., died at the Sherman Hospital, Elgin, August 24, after an illness of one year, aged 79.

William C. Hill, M.D., College of Physicians and Surgeons of Chicago, 1902, a member of the American Medical Association, and a prominent physician of Murphysboro, Ill., died at St. Andrew's Hospital, Carbondale, Ill., September 4, after an illness of five weeks, aged 25.

Edith Warner Cadwallader Crowder, M.D., Woman's Medical College of Pennsylvania, Philadelphia, 1900, formerly a member of the American Medical Association and of the Philadelphia County Medical Society, and professor of obstetrics in the Woman's Medical College of Pennsylvania, died at her home in Chicago, Aug. 18, 1906. On her marriage to Dr. Thomas R. Crowder, of Chicago, in October, 1905, all her medical appointments were resigned.

IN MEMORIAM.

Dr. W. J. Class, member of the Chicago Board of Health, died at Mercy Hospital, Benton Harbor, Michigan, Aug. 9, 1906, from cerebral thrombosis. The funeral was conducted from 151 Jansen avenue, Chi-

cago. His associates in the health department sent a floral tribute in the form of an American Medical Association membership badge. The pall bearers were Dr. C. S. Bacon, Dr. Edward Ochsner, Dr. Heman Spaulding, Dr. Henry Hartung, Dr. L. Maywith and Dr. Wm. Hessert. Dr. Class had been in ill health for some time as the result of overwork. He had recently bought a piece of land on the St. Joseph River near Benton Harbor, where he intended to build a residence. By living a quiet life, he hoped to regain his health, after which he intended to resume his scientific research work. As a result of his death, the Chicago health department lost a noted investigator and faithful inspector. Eight years ago, as a result of a civil service examination, he became a member of the department, which relation continued until the time of his death. He was a graduate of Rush Medical College, an ex-interne of Michael Reese Hospital and a postgraduate student of Vienna and Dresden. Dr. Class became prominent as a result of research work he did in connection with scarlet fever and cerebrospinal meningitis. In the first-mentioned disease, he discovered a germ which he found constantly present in over three hundred consecutive cases of scarlet fever, which germ fully complies with Koch's law. He also demonstrated through animal experiments that there is a specific antitoxic serum for this dreaded disease. His work on this germ has been published in medical journals of this and other countries. A number of years ago he published a report of a case of cerebrospinal meningitis which appeared in *The Journal* of the American Medical Association, in which he showed that the disease is contagious and that the same means should be used to prevent its spreading that are taken in other contagious diseases.

At the time of his death, he had only reached the age of 32 years. Nevertheless, as a result of untiring energy and ambition, he accomplished, within a few years, a whole life's work. Twelve years ago, when only 20 years of age, he remarked to his brother, Dr. F. L. Class, "I prefer to die at the age of 35, while having done something for mankind, than to die at a ripe old age without accomplishing anything." Dr. Class was of noble descent, descending from the house of Orleans, France. Some time in 1600, two brothers of the house of Orleans emigrated, one to Holland, while the other located in Germany, in the Province of Nassau. Dr. Class was a descendant of the German branch of the house. His paternal grandfather was very conspicuous in educational work and had great influence over those coming in contact with him. He was a brilliant writer, and the products of his pen were nearly all of them stimulated by the desire to make mankind better and happier. Many of his writings are still preserved in his native town, where they are used in teaching the youth, and when a son reaches that age at which proper guidance is essential so he will develop into a good and moral man it has become a custom in that part of Germany for the father to point to the doctor's grandfather and implore his son to pattern his life after him. Dr. Class very much resembled his grandfather, always inclining toward that which is good and pure. He was also a great admirer of Nature and good music. His father came to this country when a young

man, living with an uncle in Cincinnati, Ohio, who was in the drug business. Here he completed his pharmaceutical education, having already studied chemistry under two of Germany's most prominent chemists. He then studied medicine in Ohio Medical College and moved to Iowa, where he practiced his profession, a few years later coming to Chicago, where he engaged in the drug business, in which he continued until his death, in 1897. It was in Iowa he met and married Miss Abel, an educated and highly intellectual young lady. The result of this union was two sons, the doctor and his brother, Dr. F. L. Class, who is at present practicing in Huron, S. D. F. L. C.

CORRESPONDENCE.

Dr. Nickerson Resigns Insurance Appointment.

Dr. Nickerson has forwarded the following letter which explains itself:

Quincy, Ill., June 15, 1906.

Drs. W. R. Bross and Arthur Poll, Medical Directors Equitable Life Association, New York City, New York.

Dear Sirs:—Some time ago I received a communication from the company, stating there would be a reduction of fees from $5.00 to $3.00 for examinations. This I do not consider adequate compensation for the service. I have been your chief examiner at Quincy, Ill., and nominator for the ten adjacent counties the past twenty years. The company has always treated me handsomely, and I have no reason to complain of past favors. The Illinois State and Adams County Medical Societies recently passed resolutions asking their members to desist from making examinations on the basis of this reduction. Being a member of these societies, I will have to present my resignation as your examiner and nominator.

Yours respectfully,

L. H. A. Nickerson, M.D.

NEW MEMBERS OF THE AMERICAN MEDICAL ASSOCIATION.

The following new members from Illinois have been received during the last three months:

JULY.

Amerison, G. C., Chicago.
Barnes, H. T. Maywood.
Brown, A. L., Vienna.
Buckley, J. E., Vienna.
Burnside, L. A., Martinsville.
Camerer, J. D., Kinmundy.
Carpenter, F. E., Chicago.
Catlin, S. R., Rockford.
David, F. E., Chicago.
Diamond, P. T., Chicago.
Eakin, A. C., Shirland.
Egan, J. C., Chicago.
Engels, N. R., Chicago.
Ferguson, Clara, Chicago.
Foelsch, A. J., Bondville.
Fox, C. M., Chicago.
Freeman, H. E., Millington.
French, W. M., Chicago.
Fults, J. C., Waterloo.
Gleeson, Benj., Danville.
Hammond, J. L., Chicago.
Hecox, C. R., Golden.
Hubert, J. F., Chicago.
Hyde, D. L., Chicago.
Jefferson, F. A., Chicago.
Lyness, J. D., Savannah.
McDavid, J. T., Decatur.
Nickerson, L. H. A., Quincy.
Orcutt, D. C., Chicago.
Ostrum, Louis, Rock Island.

Patterson, H. A., Joliet.
Selby, F. S., Chicago.
Smith, S. A., Annapolis.
Stookey, V. P., Colchester.
Tarr, A. W., Grand Chain.
Thomas, H. B., Chicago.
Waggoner, E. L., Lebanon.
Weber, F. J., Olney.
Williams, H. O., Belknap.

AUGUST.

Baker, R. L., Peoria.
Blatt, M. L., Chicago.
Dart, Ralph, Rock Island.
Ellis, I. W., Murphysboro.
Evans, C. A., Bluffs.
Griffin, M. A., Chicago.
Hobson, E. B., Gillespie.
Hoover, W. K., Lovington.
Kilbourn, E. D., Ancona.
Koch, J. A., Quincy.
Lence, J. J., Jonesboro.
McGinnis, Edwin, Orland.
McKee, H. T., Sparta.
Morgan, J. W., Moline.
Pautler, N. B., Waterloo.
Post, W. E., Chicago.
Roe, M. C., Chana.
Rose, W. J., Columbia.
Schmidt, H. G. G., Elgin.
Schrayer, W. S., Chicago.
Smith, C. G., Red Bud.
Stewart, W. B., Joliet.
Taylor, L. S., Elgin.
Wharton, J. E., Jacksonville.
Xelowski, T. Z., Chicago.

SEPTEMBER.

Babb, Helen, Springfield.
Baxter, A. J., Astoria.
Benson, E. O., Chicago.
Cloud, M. Morgan, Chicago.
Colbourne, J. A., Pontiac.
Corbus, Howard, Kankakee.
Colby, Chas. P., Springfield.
Combs, Geo. W., Ridgeway.
Counett, James E., Lawrenceville.
Culhane, T. H., Rockford.
Dinger, H. A., Red Bud.
Davisson, Alfred W., Peoria.
Ede-Punte, Janet J., Chicago.
Ferry, Lemuel A., Geneseo.
Gerould, T. F., Centralia.
Hager, Daniel S., Chicago.
Hassett, James J., McLeansboro.
Heintz, Edw. Louis, Chicago.
Horme, Elmer S., Colfax.
Hubbard, C. M., Virginia.
Huston, J. W., Virginia.
Kelly, J. W., Springfield.
Kraft, O. H., Chicago.
Lovewell, C. Hubert, Chicago.
Morgan, Fred B., Cornell.
Morgan, F. R., Towanda.
Quist, Frank J., Chicago.
Rawlins, Benj. L., Chicago.
Ritchey, R. M., Menard.
Stotz, Charles Frederick, Chicago.
Songer, Walter E., Menard.
Taylor, John F., Buda.
Webb, William F., Chicago.

NEW MEMBERS OF ILLINOIS STATE MEDICAL SOCIETY.

During the months of July and August the following physicians have become members of the Illinois State Medical Society:

JULY.

E. M. Minnick, Moline, Rock Island County.
B. C. Anderson, Rockford, Winnebago County.
H. M. Voris, East St. Louis, St. Clair County.
John T. Higgs, East St. Louis, St. Clair County.
A. B. McQuillan, East St. Louis, St. Clair County.
Louis J. Linder, East St. Louis, St. Clair County.
T. C. Keisling, Lebanon, St. Clair County.
J. C. Caldwell, East St. Louis, St. Clair County.
Chas. S. Skaggs, East St. Louis, St. Clair County.
L. T. Milles, Caseyville, St. Clair County.
F. L. Evans, East St. Louis, St. Clair County.
Otto W. Knuvitz, East St. Louis, St. Clair County.
W. F. Johnson, Ridgeway, St. Clair County.
W. H. Riley, Ridgeway, St. Clair County.
Claud Sanders, Irvington, Montgomery County.
H. M. Glass, East St. Louis, St. Clair County.
H. M. Daly, Pontiac, Livingston County.
S. M. Barnes, Pontiac, Livingston County.

AUGUST 1.

J. G. Ellis, Cerrogordo, Piatt County.
Eugene Coffin, Peoria, Piat County.
F. F. Davis, Jeffersonville, Wayne County.

W. A. Bulany, Keens, Wayne County.
F. G. Bufour, Oquawka, Henderson County.
C. B. Caldwell, Monticello, Piatt County.
G. T. Cremeens, Dahlgren, Hamilton County.
David C. English, New Brunswick, New Jersey County.
V. S. Fildes, Parkersburg, Richland County.
E. C. Ferguson, Edwardsville, Madison County.
P. M. Gray, Cisne, Wayne County.
H. L. Powell, Bloomington, McLean County.
Charles Jones, Noble, Richland County.
G. A. Kelson, Orchardville, Wayne County.
R. B. Smith, Orchardville, Wayne County.
George Steely, Danville, Vermillion County.
A. F. Turner, Taylorville, Christian County.
J. M. Threadgill, New Douglas, Madison County.
J. L. Young, Rinard, Wayne County.
A. L. Ward, Bement, Piatt County.
C. E. Johnson, Johnsonville, Wayne County.
E. S. Herriott, Jacksonville, Morgan County.
Homer A. Millard, Minonk, Woodford County.
L. H. Wiman, LaMoile, Bureau County.
C. H. Miller, Boody, Macon County.
L. N. Lindsay, Forsyth, Macon County.

NEW BOOKS.

SURGICAL SUGGESTIONS. By Drs. Brickner and Moschocowitz. Price, 50 cents, postpaid.

This book is novel, not only on account of the many terse and epigrammatic suggestions given, but because of its general appearance and attractive form. It contains 250 suggestions, grouped under proper headings, and its contents are carefully indexed. While some of the items are familiar to the practiced surgeon, they are all presented in a manner that will impress them on the reader's memory. The book is bound in heavy cloth, stamped in gold, and the text is printed upon India tint paper, with marginal headings in red. This book will be much appreciated by the general practitioner, not alone on account of the value of its contents, but also as an artistic bit of book-making.

PROPHYLAXIS AND TREATMENT OF INTERNAL DISEASES. By Frederick Forchheimer, M.D., Professor of Theory and Practice of Medicine and Clinical Medicine, Medical College of Ohio, University of Cincinnati, Cincinnati, Ohio. Cloth. Price, $5.00 net.

This is an eminently practical work, one which concerns itself diligently with the business in hand. It first lays down broad principles, then details the special application of them, where possible; failing that, it indicates the proper direction for their application.

It is essentially a work of breadth. It is also essentially a work of experience. Free from dogmatism, there is the calm assurance of one to whom the path is familiar. In these days of therapeutic pessimism, it is refreshing to find a practical physician to whom the making of a correct diagnosis is but the beginning rather than the end of his craft.

Dr. Forchheimer undertook a difficult task, but he has given us a most excellent work, one that will have a large sale to the general practitioner, a book which has long been in demand.

A TEXT-BOOK OF PHYSIOLOGY. By J. R. Murlin, Ph.D., Instructor of Physiology, Bellevue Medical College, New York City. With an introduction by Graham Lusk, Ph.D., Professor of Physiology, Bellevue Medical College. This is a translation of Tigerstedt's Physiology.

Aside from the general excellence of this book it presents a number of noteworthy and commendable features. The first of these is the manner of approach to the real subject. After a chapter on General Method, in which the author gives some instances of how exact physiological knowledge is gained, he lays a very broad foundation to the special subject of human physiology. The second chapter is on the Physiology of the Cell. This constitutes the biological foundation. The next is a chapter on the Chemical Constituents of the Body, which supplies the chemical foundations. The fourth chapter is devoted to Metabolism and Nutrition. Every chapter of the book is as complete as possible within the limits of a single

volume, and many of them are very originally conceived. This is especially true of the chapter on Metabolism and Nutrition. The chapter on the Circulation and the one on Metabolism and Nutrition fall within the author's own chosen fields of investigation. Needless to say they contain much that is new and of great importance. In his treatment of the Physiology of the Nervous System the author reaches the climax of the book, whether we consider it from the standpoint of the practitioner of medicine or of the general reader. We should not neglect to mention the great number of excellent illustrations. There are 305 illustrations in this work, 63 of which are in colors.

SURGICAL PATHOLOGY AND TREATMENT OF DISEASES OF THE EAR. By Clarence John Blake, Professor of Otology in Harvard University, and Henry Ottridge Reik, Associate in Ophthalmology and Otology, Johns Hopkins University. The price of this work, in cloth binding, is $3.50.

This work begins with a chapter on Surgical Anatomy of the Temporal Bone and Adnexa, following which is a chapter on Aseptic Technic; Chapter III, Diseases of the Auricle and External Auditory Canal; Chapter IV, Diseases of the Tympanic Membrane and Tympanum; Chapter V, The Possible Complications and Consequences of Suppurative Otitis Media; Chapter VI, Middle Ear Operations; Chapter VII, Mastoid Operations; Chapter VIII, Adventitious Aural Surgery. This chapter treats of a considerable number of operations, which, though not strictly a part of otology, are, however, necessarily connected with this work: Adenoids, their effect on the organ of hearing; adenoidectomy, subcutaneous and intravenous infusions, lumbar puncture.

The book is well illustrated, mostly from original drawings. A few, however, are copies from original drawings made for Professor Politzer and placed at the author's disposal.

ILLINOIS STATE MEDICAL SOCIETY

MEDICO-LEGAL COMMITTEE.

EXECUTIVE COMMITTEE.

FROM ILLINOIS MEDICAL SOCIETY.

W. A. Evans, 103 State St., Chicago. Central 2740.
H. N. Moyer, 103 State St., Chicago. Central 2751.
C. D. Pence, 859 Turner Ave., Chicago. Canal 1335.
E. W. Wels, Ottawa, Ill.
M. L. Winstead, Wetaug, Ill.

FROM CHICAGO HOMEOPATHIC MEDICAL SOCIETY.

N. B. Delameter, 31 Washington St., Chicago. Central 1926.
J. B. Cobb, 42 Madison St., Chicago. Central 32.

GENERAL COUNSEL.

Calhoun, Lyford & Sheean, 806 The Rookery, Chicago.

This list shows the names of members of the medicolegal committee reported to the chairman up to the time of going to press. County societies are requested to report any changes or additions at once:

COUNTY.	REPRESENTATIVE.	ADDRESS.
Adams	John O. Koch	Quincy
Alexander		
Bond		
Boone		
Brown	William Parker	Mt. Sterling
Bureau		
Calhoun		
Carroll	G. W. Johnson	Savanna
Cass	John A. Glenn	Ashland
Cook County, Chicago Medical Society	W. A. Evans	103 State St., Chicago
	H. N. Moyer	103 State St., Chicago
	C. D. Pence	1389 Ogden Ave., Chicago
Champaign	H. E. Cushing	Champaign
Christian	J. N. Nelms	Taylorville
Clark	R. H. Bradley	Marshall
Clay	E. P. Gibson	Louisville

COUNTY.	REPRESENTATIVE.	ADDRESS.
Clinton	J. J. Moroney	Breese
Coles	I. T. Montgomery	Charleston
Crawford	I. L. Firebaugh	Robinson
Cumberland	W. R. Rhodes	Toledo
De Kalb	G. W. Nesbitt	Sycamore
De Witt	G. S. Edmondson	Clinton
Douglas	W. S. Martin	Tuscola
Edgar		
Edwards	C. S. Brannan	Albion
Effingham		
Fayette	E. W. Brooks	St. Elmo
Franklin		
Fulton		
Gallatin	T. Alfred Jones	Ridgeway
Greene	H. A. Chapin	White Hall
Grundy	H. M. Ferguson	Morris
Hamilton	Henry E. Hale	McLeansboro
Hancock	Charles L. Ferris	Carthage
Hardin	J. A. Wernack	Karbers Ridge
Henderson	J. P. Riggs	Media
Henry	C. W. Hall	Kewanee
Iroquois-Ford	O. O. Hall	Milford
Jackson	J. T. McAnally	Carbondale
Jasper	James P. Prestly	Newton
Jefferson	J. H. Mitchell	Mt. Vernon
Jersey	John S. Williams	Jerseyville
Jo Daviess	D. G. Smith	Elizabeth
Johnson		
Kane-McHenry	Geo. F. Allen	Aurora
Kankakee		
Knox	Ben. D. Baird	Galesburg
Lake		
La Salle	E. W. Weis	Ottawa
Lawrence	B. F. Hochman	Sumner
Lee	R. B. Owens	Dixon
Livingston		
Logan	Carl Rembe	Lincoln
McDonough		
McLean	E. Mammen	Bloomington
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Madison	J. N. Shaff	Alton
Marion	W. D. Richardson	Centralia
Marshall	S. O. Hendricks	Henry
Mason		
Massac	A. C. Ragsdale	Metropolis
Menard	Burton W. Hole	Tallula
Mercer	M. G. Reynolds	Aledo
Monroe		
Montgomery		
Morgan	J. N. Hairgrove	Jacksonville
Moultrie		
Ogle	F. N. Mitchell	Leaf River
Peoria	L. A. McFadden	Peoria
Perry		
Platt	C. M. Bumstead	Monticello
Pike	L. J. Harvey	Griggsville
Pope		
Pulaski	M. L. Winstead	Wetaug
Putman		
Randolph	H. C. Adderly	Chester
Richland		
Rock Island	G. L. Eyster	Rock Island
St. Clair	C. E. Auten	Belleville
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Sangamon	B. B. Griffith	Springfield
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Shelby	Frank Auld	Shelbyville
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Warren	F. E. Wallace	Monmouth
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Wayne		
Whiteside	Charles G. Beard	Sterling
White	B. S. Crebs	Carmi
Will	Frank C. Fisher	Joliet
Williamson		
Winnebago	Charles Winn	Rockford
Woodford		